



IN THE NAME OF GOD

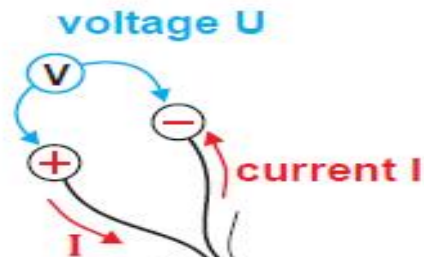
PACE MAKER AND ICDs

Dr.Sima Sayah

Local effects of cardiac electrical stimulation

- Cardiac pacing requires a local stimulus sufficient to depolarize local myocardium during diastole to initiate a self-propagating wave front of depolarization.
- A stimulus that successfully stimulates local myocardium is said to capture it.

PACING IMPEDANCE

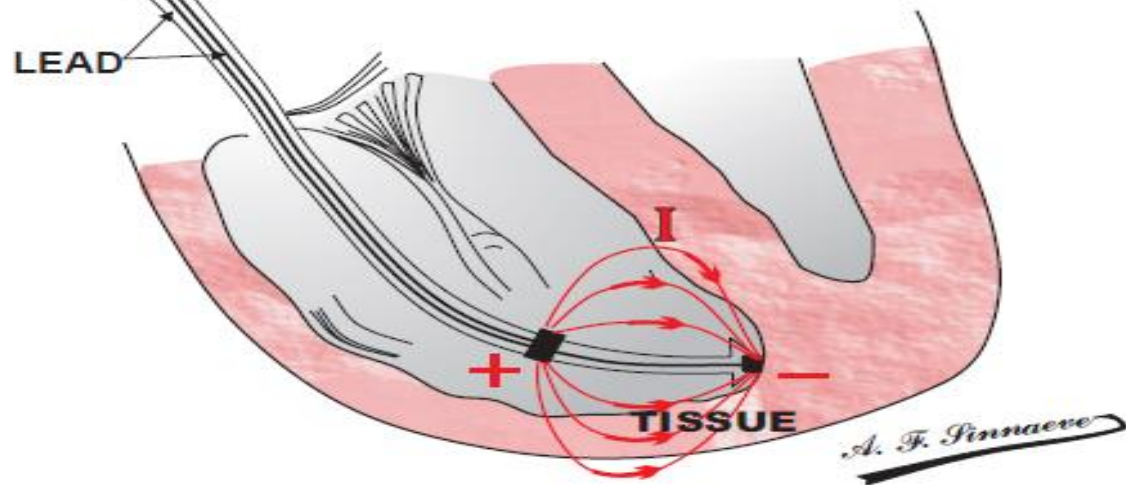


According to Ohm's law :

$$R = \frac{\text{VOLTAGE } U \text{ (in volt)}}{\text{CURRENT } I \text{ (in ampere)}}$$

comprises :

- * lead resistance
- * tissue impedance



INSULATION
DEFECT
< 250Ω

NORMAL
PACING
IMPEDANCE
ca. 500Ω

LEAD
FRACTURE
> 1000Ω



Note for electricians :

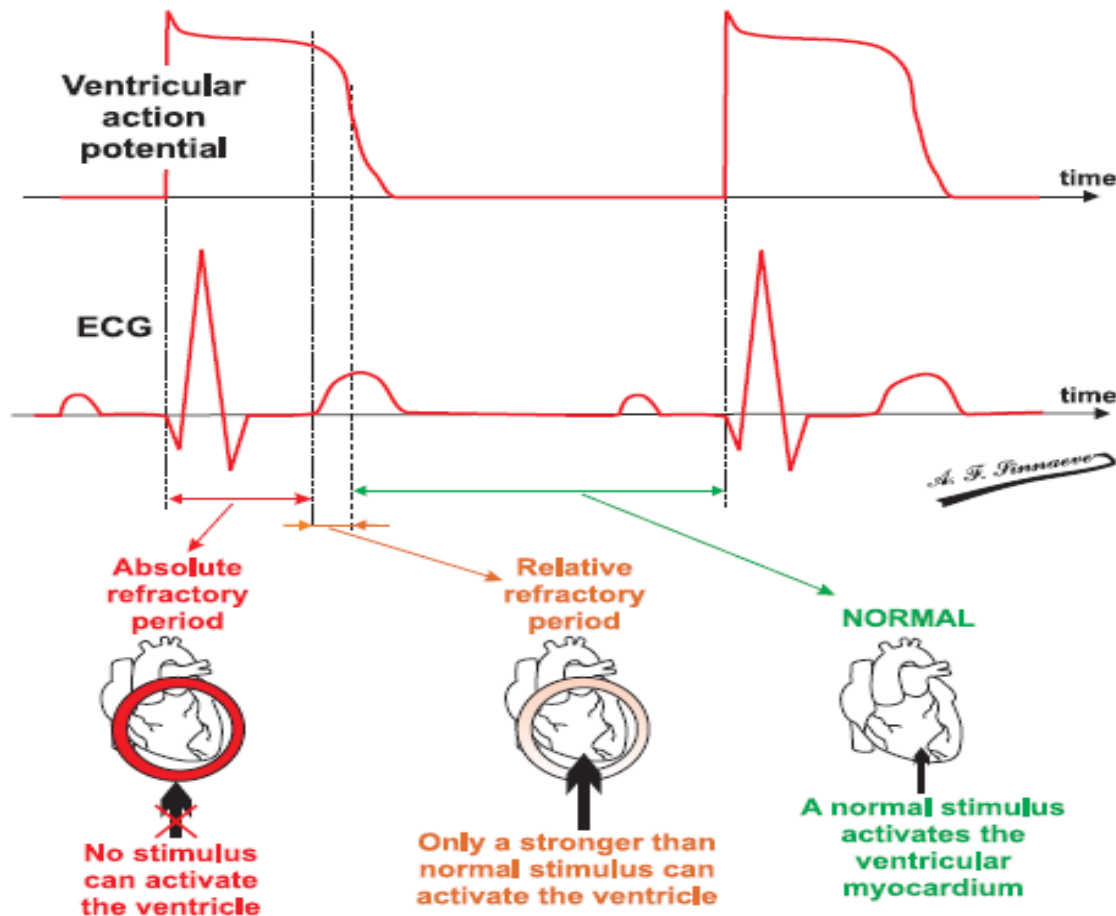
The pacing impedance is not purely resistive (the tissue impedance is capacitive) and should be indicated by a Z . In the clinical practice only the absolute value or magnitude of the pacing impedance is considered and since it is expressed in "OHM" according to Ohm's law, most people simply call it "resistance"

- The stimulus strength for local capture by ATP is higher than that for bradycardia pacing because ATP pulses are delivered during the relative refractory period rather than during diastole.



Always keep in mind ! The myocardial refractory period refers to stimulation. In contrast the pace-maker refractory period refers to the sensing function of the device.

VENTRICULAR MYOCARDIAL REFRACTORY PERIOD

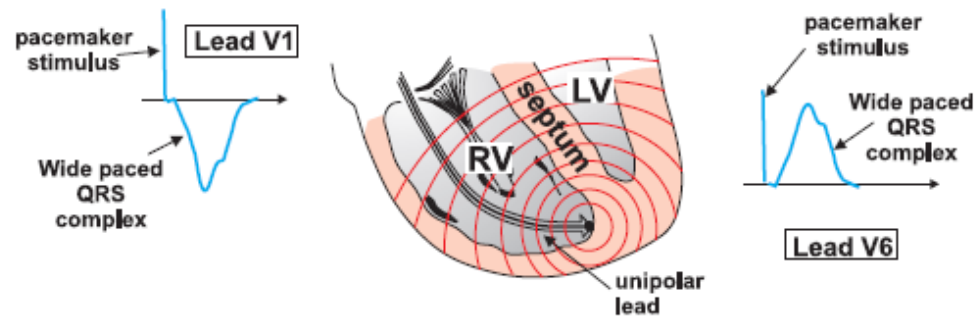


VENTRICULAR DEPOLARIZATION BY PACING

- * The depolarization caused by the pacemaker does not occur via the specialized His-Purkinje network and propagates slower through ordinary myocardium
- * The QRS complex is therefore wide like a ventricular extrasystole (premature ventricular contraction)

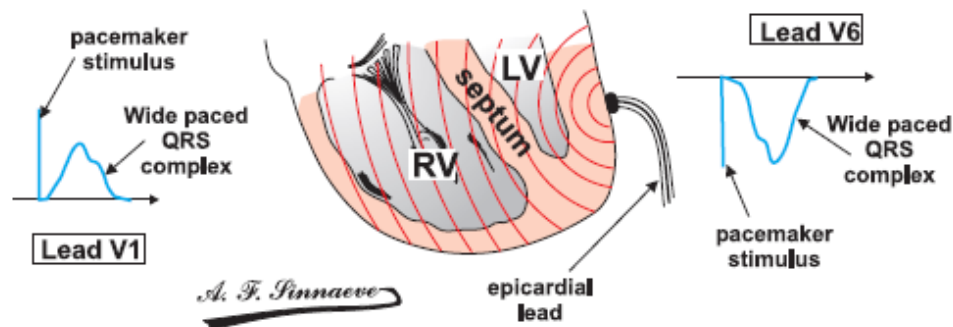
ENDOCARDIAL STIMULATION FROM RIGHT VENTRICLE

ECG resembles LBBB (LBBB = left bundle branch block)

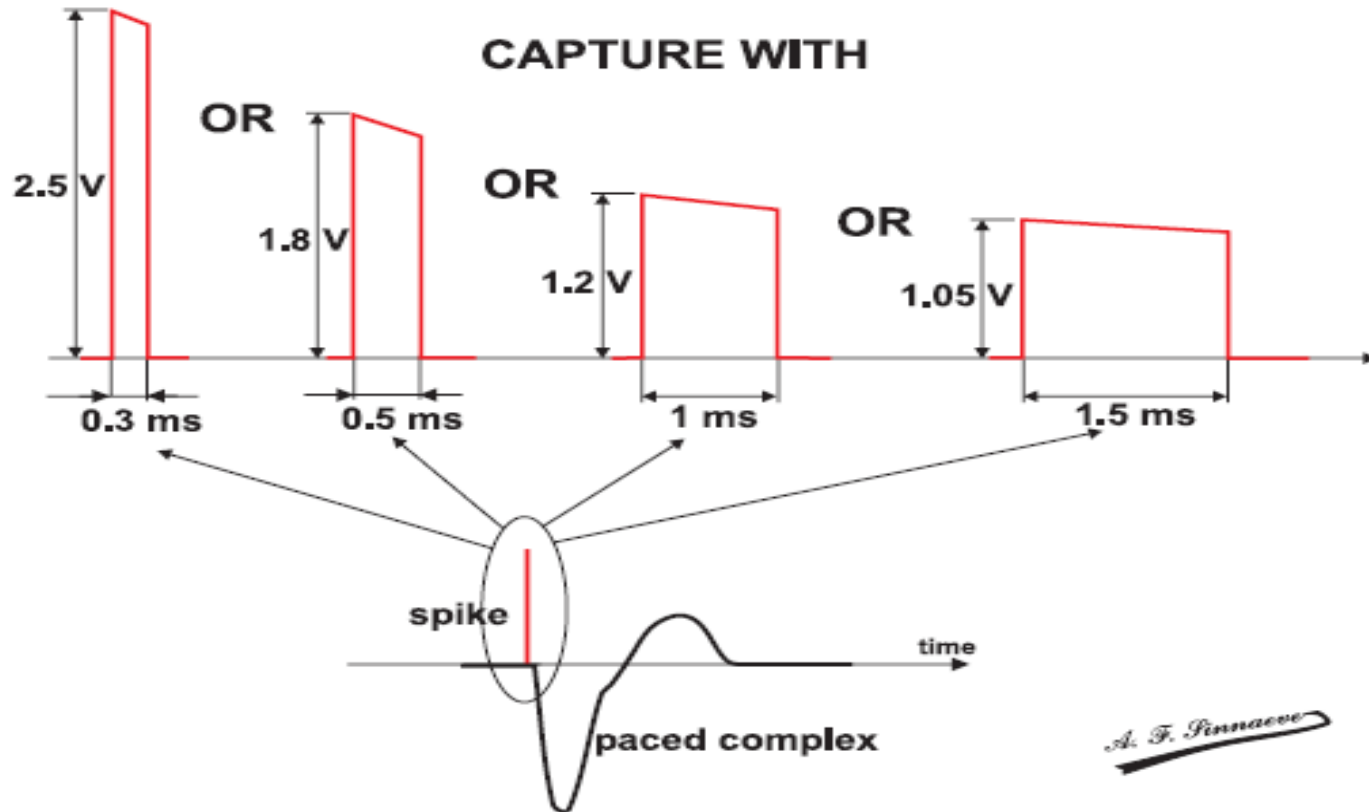
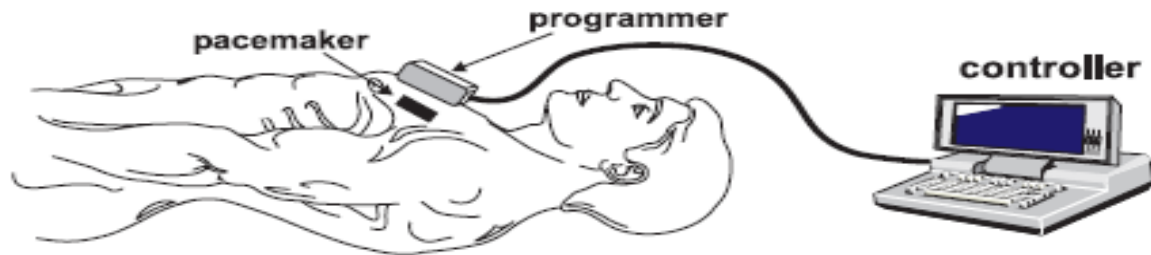


EPICARDIAL STIMULATION FROM LEFT VENTRICLE

ECG resembles RBBB (RBBB = right bundle branch block)



VOLTAGE AND PULSE DURATION MAY BE CHANGED BY AN EXTERNAL PROGRAMMER

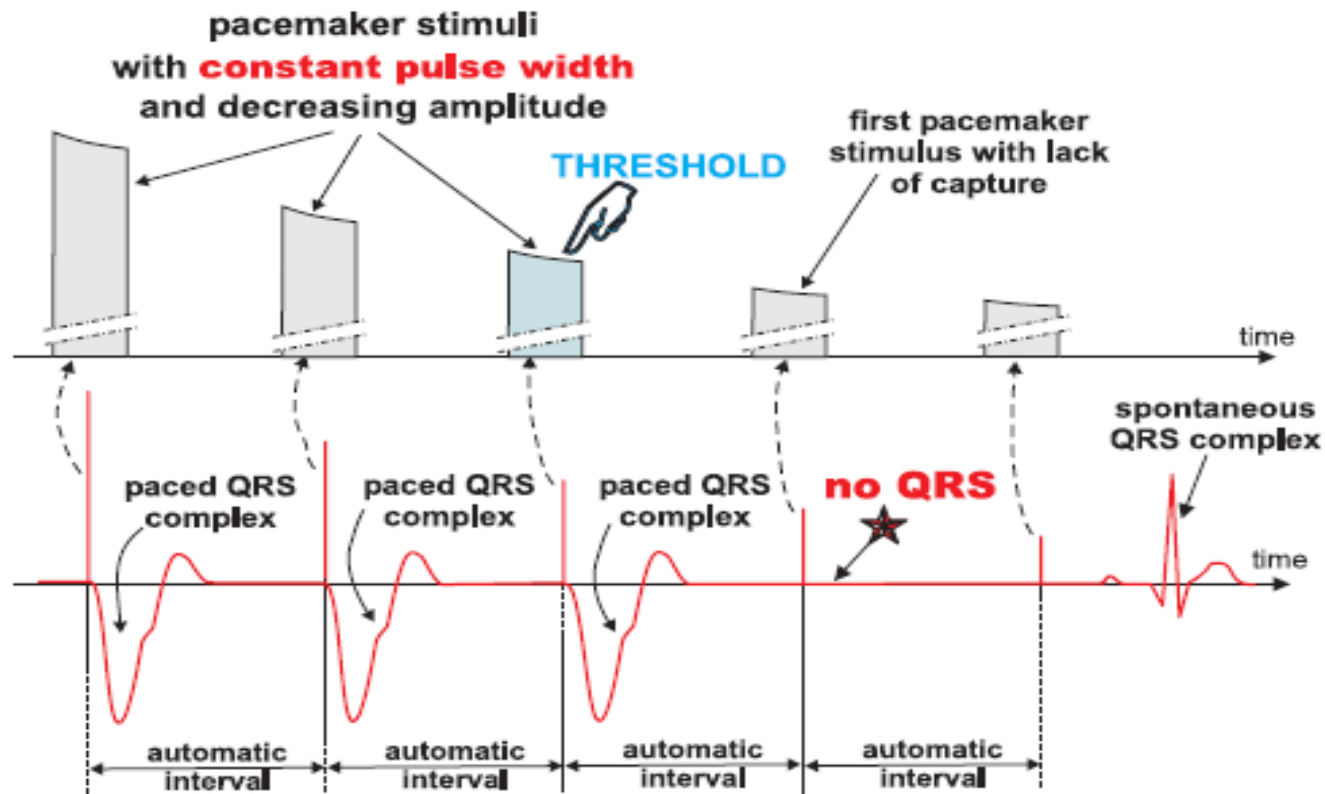


A. F. Pinnaeve

Threshold for pacing and defibrillation

- A threshold stimulus is the minimum stimulus required to evoke a response.(to depolarize local myocardium and to initiate a propagated response)
- The term defirilation threshold(DFT) is used as the minimum shock strength that defibrillates during a sequence of fibrillation episodes in which defibrillation test shocks of different strengths are delivered

DETERMINATION OF PACING THRESHOLD



A. F. Pinnaone



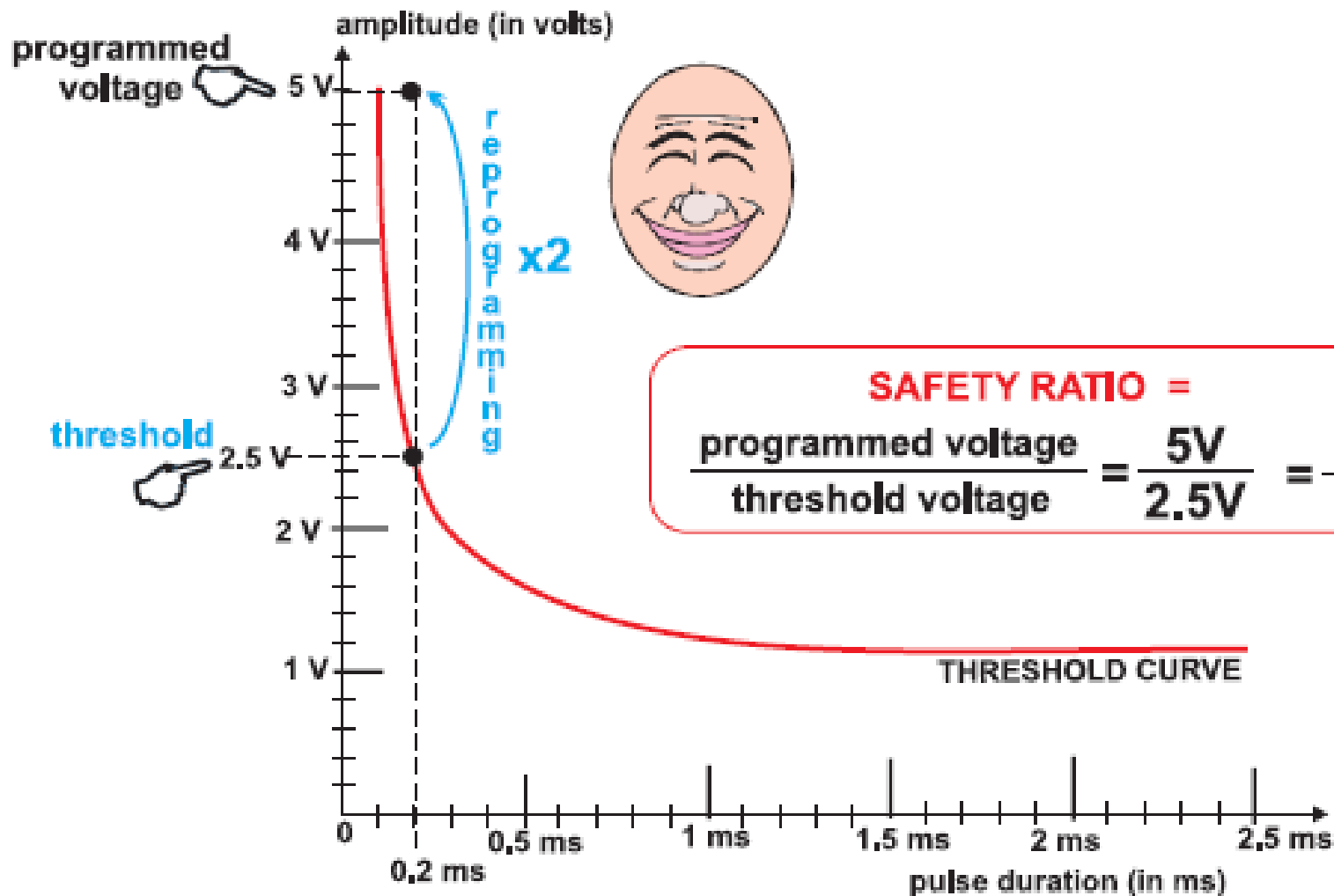
REMINDER

The pacing threshold is always expressed in terms of both voltage and pulse duration. The pacing threshold can be determined in terms of the smallest output voltage that captures the heart while keeping the pulse duration constant.

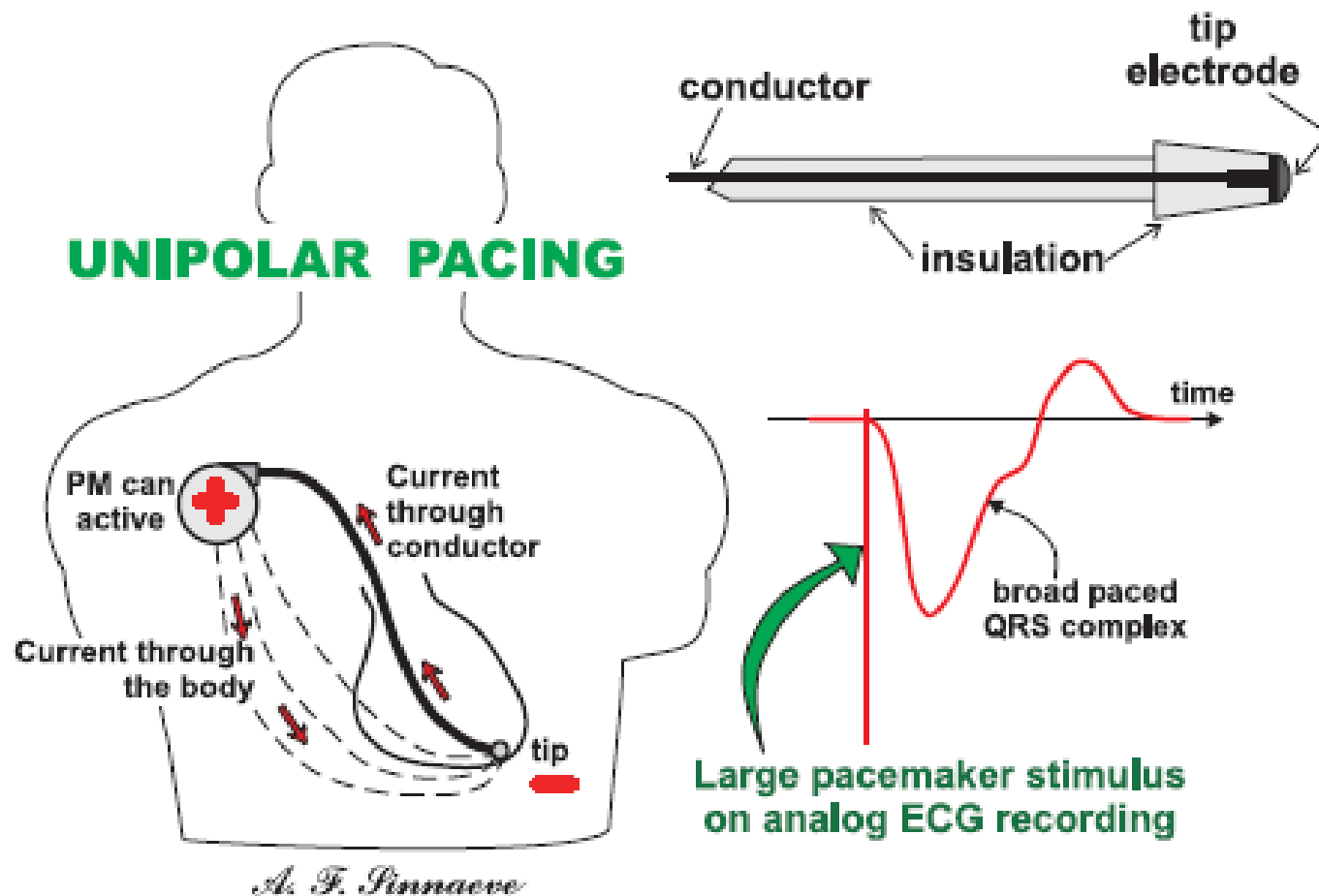
Waveform duration is critical

- Typically, the voltage output for pacing is set to 1.5-2 times the threshold at pulse durations of 0.4 -0.5 ms.

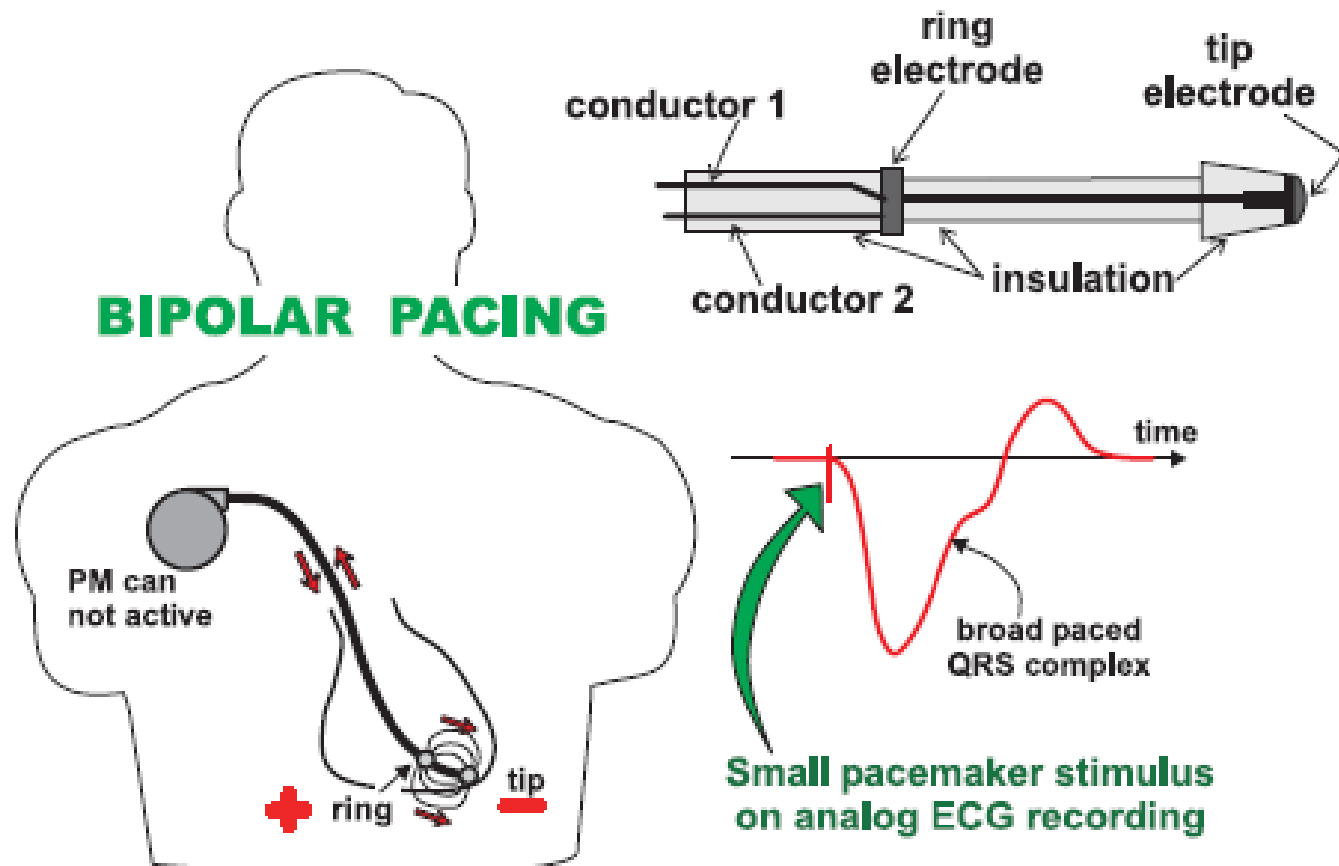
SAFETY RATIO CONCEPT FOR CAPTURE



BIPOLAR vs UNIPOLAR PACING SIZE OF THE STIMULUS ON ECG

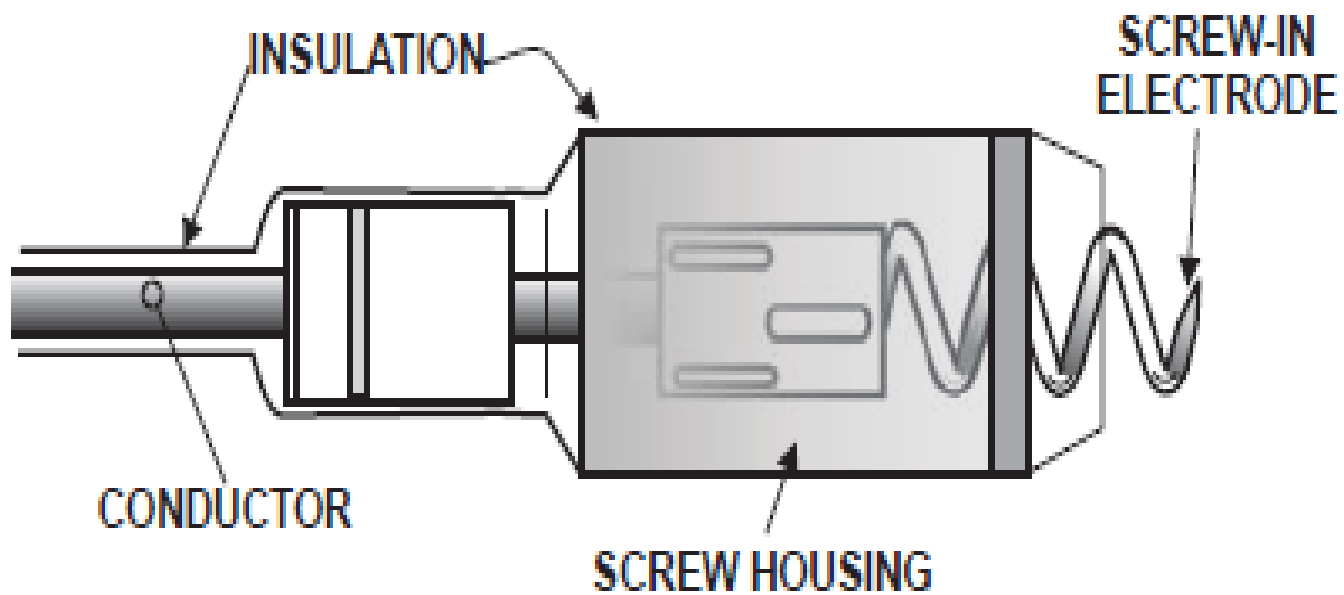


BIPOLAR vs UNIPOLAR PACING SIZE OF THE STIMULUS ON ECG



A. F. Pinnaeve

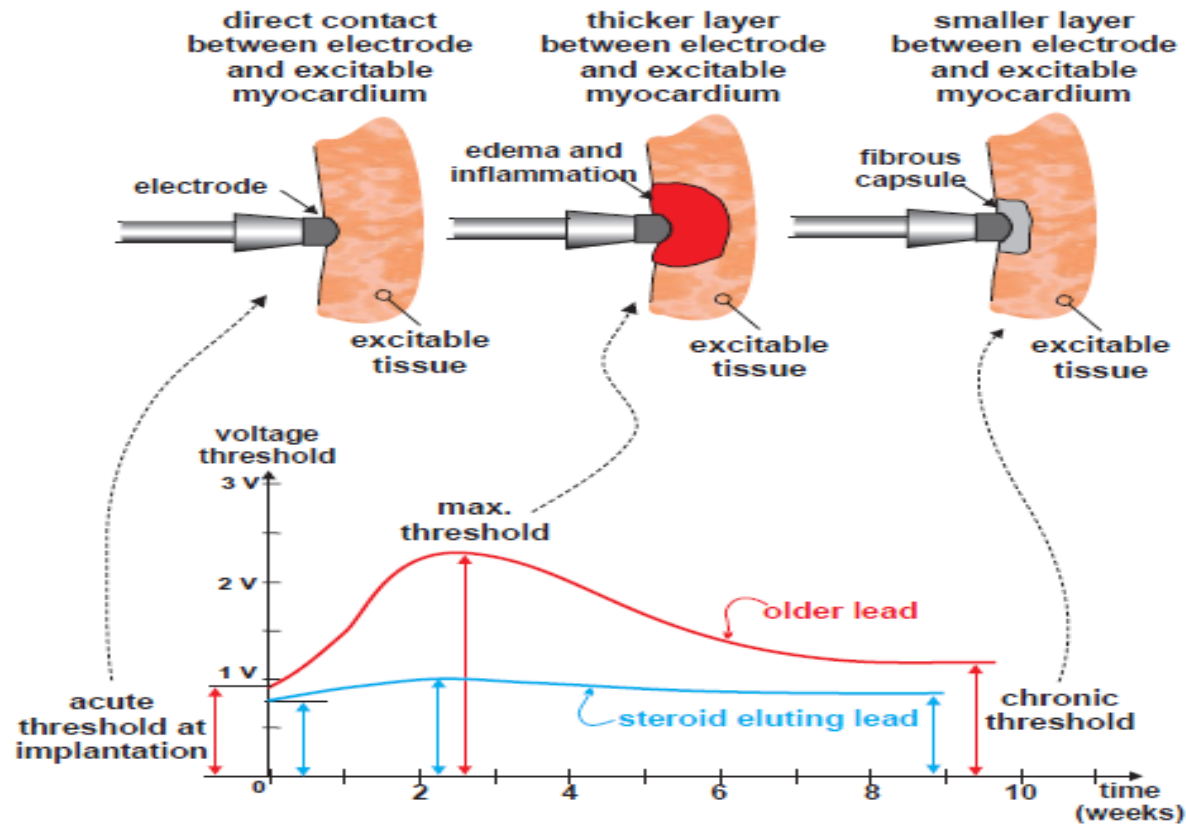
ACTIVE LEAD FIXATION



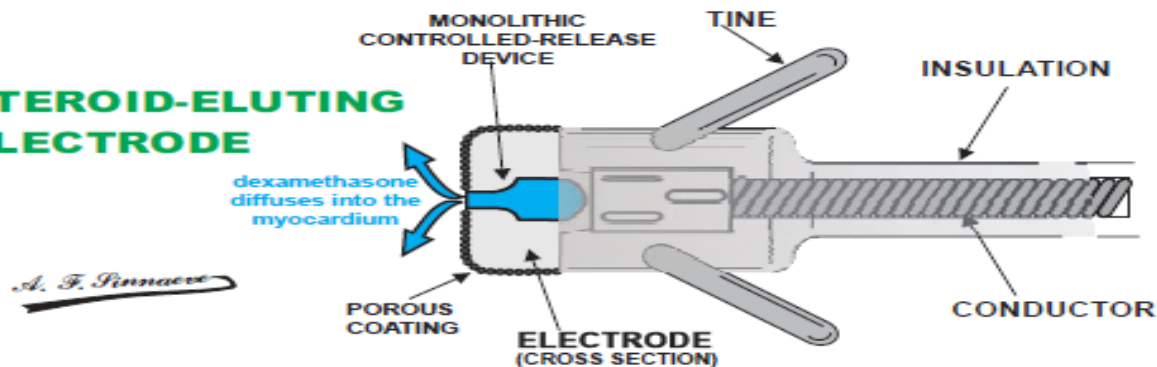
Voltage is a critical parameter for pacing or defibrillation because it determines the electrical field that interacts with the heart

- $E=IR$
- E: voltage
- I:current
- R:resistance

EVOLUTION OF PACING THRESHOLD



STERIOD-ELUTING ELECTRODE





THREE-LETTER PACEMAKER CODE (ICHD)

POSITION	1st	2nd	3rd
CATEGORY	CHAMBER(S) PACED	CHAMBER(S) SENSED	MODE OF RESPONSE
LETTERS	V = VENTRICLE A = ATRIUM S = SINGLE	V = VENTRICLE A = ATRIUM S = SINGLE O = NONE	T = TRIGGERED I = INHIBITED O = NONE

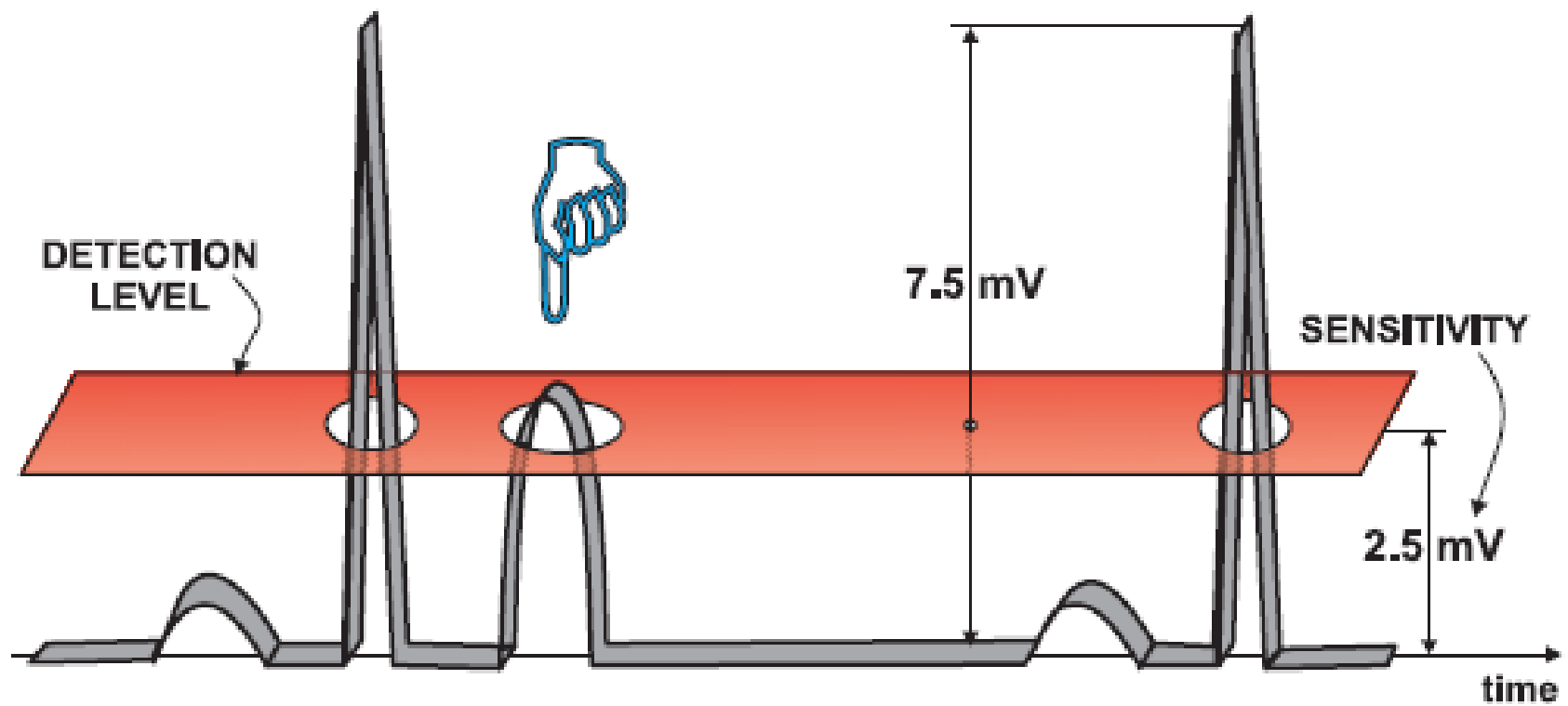
EXAMPLES :

AAI = a pacemaker pacing and sensing in the atrium, being inhibited by spontaneous electrical activation of the atrium

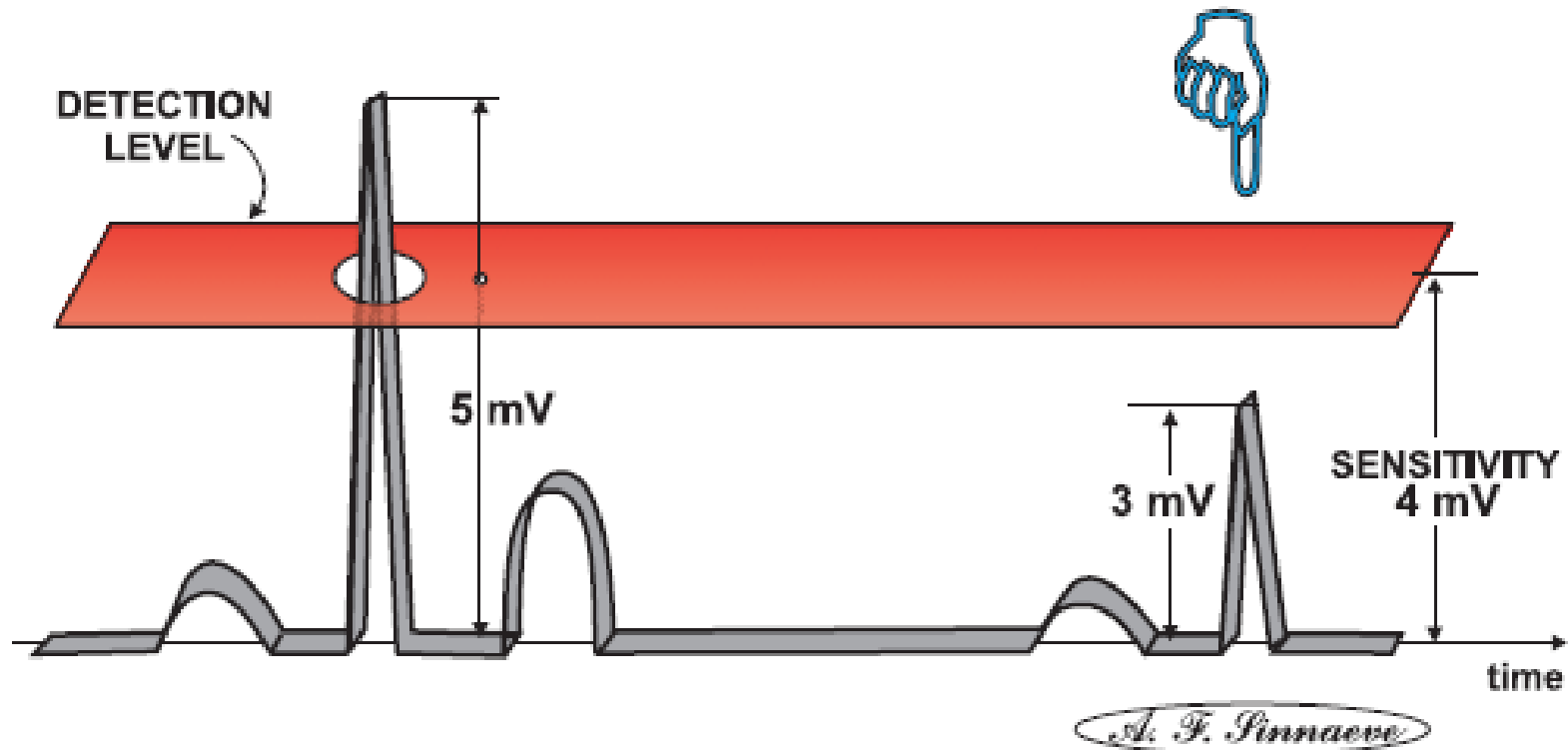
VVT = a pacemaker pacing and sensing in the ventricle and working in the triggered mode (each sensed ventricular event elicits a pacemaker stimulus)

A. F. Pinnaeve

OVERSENSISING



UNDERSENSING



The voltage refers to the intracardiac electrogram and not the surface QRS complex. Sensitivity refers to a programmable parameter of the pacemaker. A sensitivity of 4 mV means that the pacemaker can only sense a signal equal to or greater than 4 mV. It will sense a signal of 5 mV but not a signal of 3 mV.

Various drugs and metabolic effects can alter pacing and defibrillation thresholds

- ❑ -acidosis
- ❑ -alkalosis
- ❑ Electrolyte abnormalities ,especially hyperkalemia
- ❑ -drugs: 1C(flecainide),amiodaron

Goals of pacing:

- ❑ 1. Restoration of rate responsiveness
- ❑ 2. Restoration of AV synchrony

AV synchrony

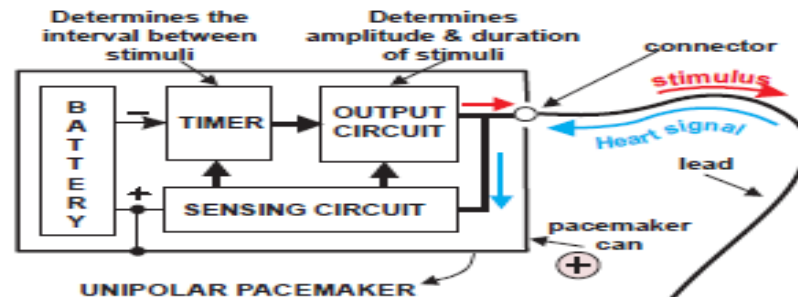
- In patients with CMP & HF, timing of RV and LV contraction and relaxation may differ sufficiently that optimal AV timing for one may not be ideal for the other.
- Patients with severe diastolic dysfunction may benefit most from AV synchrony because they depend on optimal preload.

- ❑ Lack of AV synchrony as a result of exclusive ventricular pacing can result in hemodynamic impairment caused by VA conduction and atrial contraction against a closed AV valve.
- ❑ sign's and symptoms
:malaise,lightheaded ness ,atypical chest dyscomfort
- ❑ (pace maker syndrom)

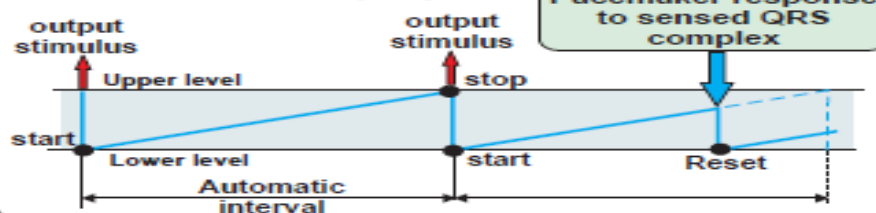
- ❑

VENTRICULAR DEMAND PACING (VVI)

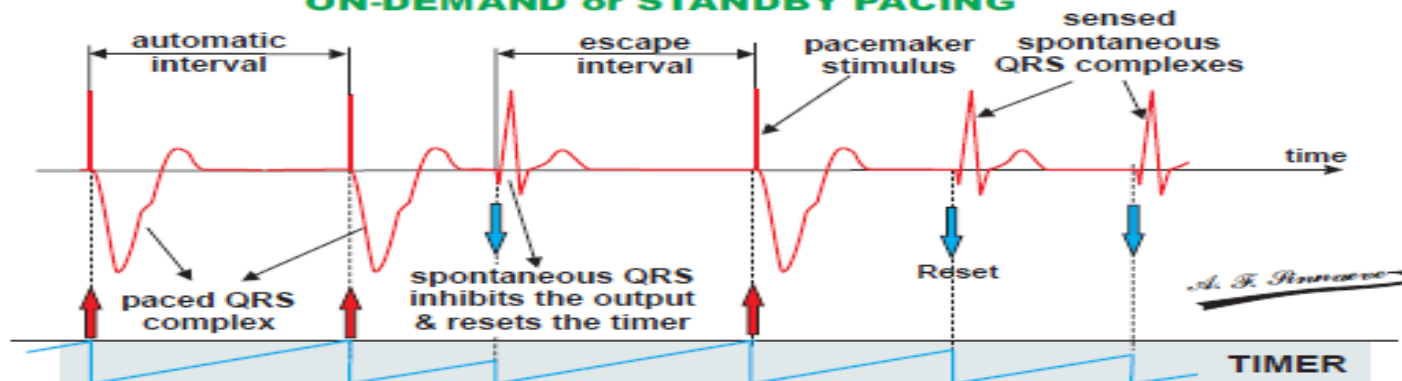
Note that in the VVI mode, a competitive rhythm is not possible. Moreover the lifetime of the battery is extended because the pacemaker is not pacing during long periods of time when it is on standby.



TIMER ACTION

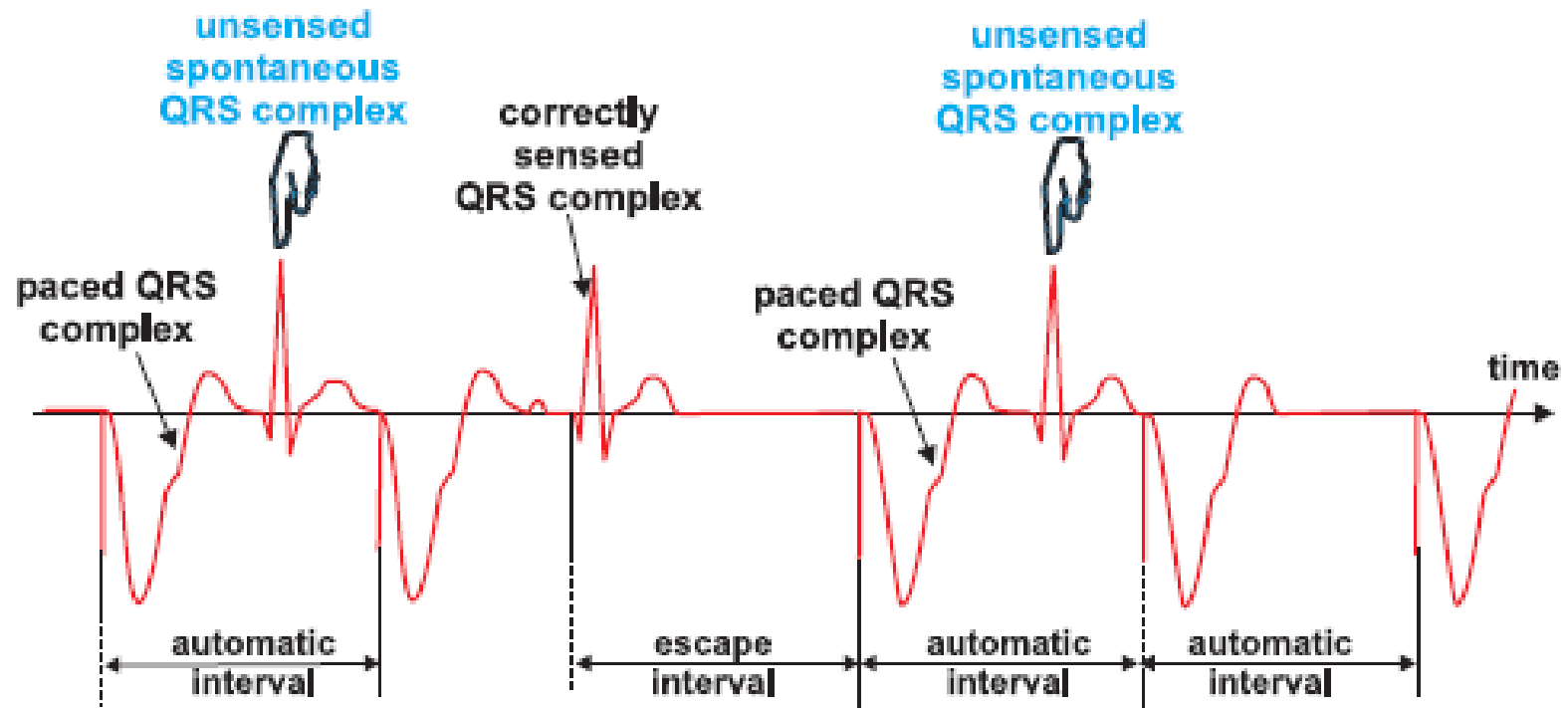


ON-DEMAND or STANDBY PACING



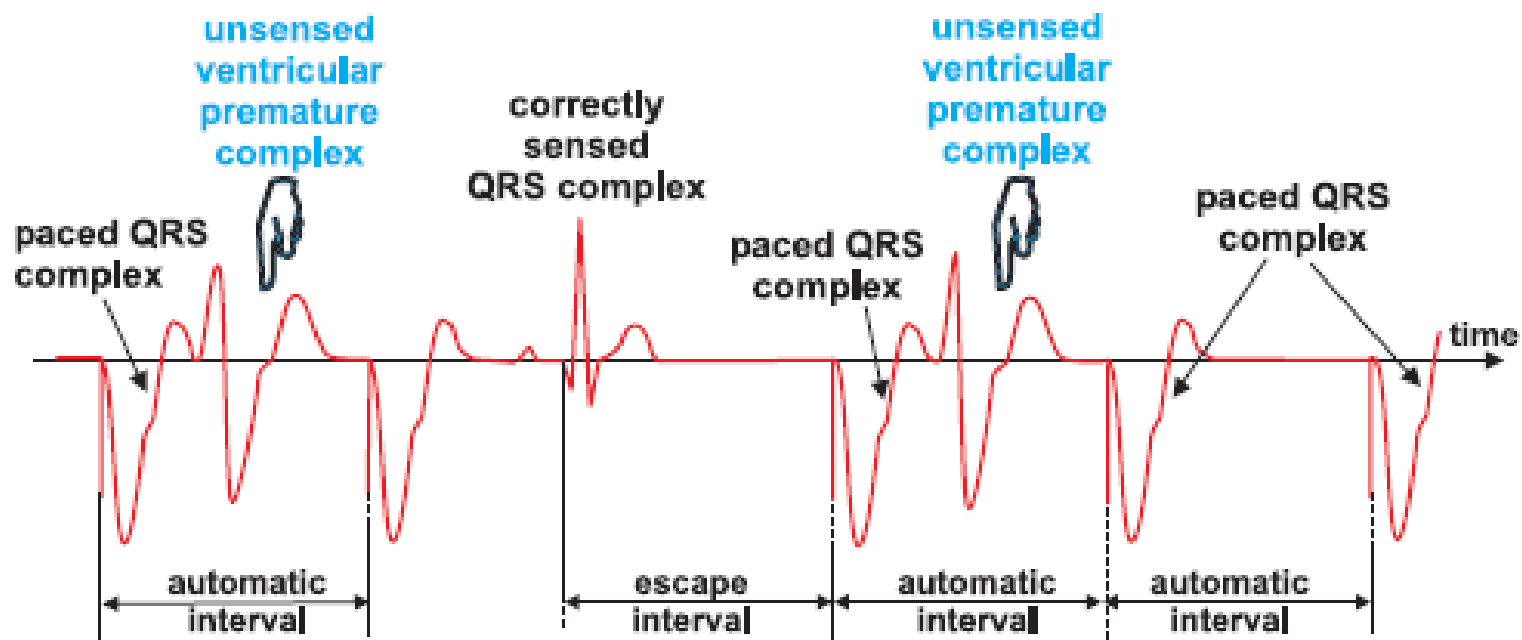
The electronic escape interval (starting at the time of intracardiac sensing) is equal to the automatic interval. The escape interval is measured on the surface ECG from the onset of the QRS complex because the time of intracardiac sensing cannot be determined accurately. Therefore the escape interval so measured will be slightly longer than the automatic interval because intracardiac sensing occurs later than the beginning of the surface QRS complex.

1 INTERMITTENT UNDERSENSING OF A DEMAND PACEMAKER



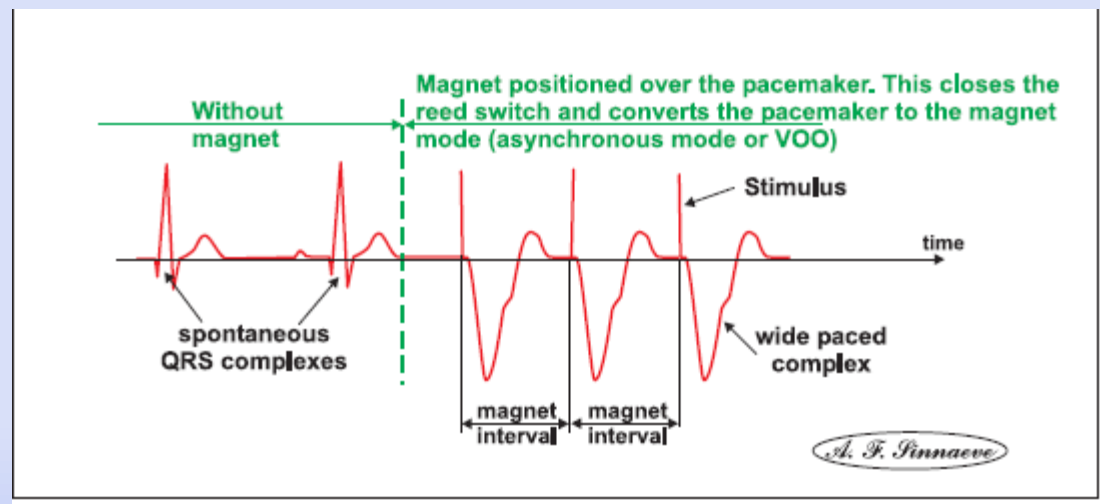
2

UNDERSENSING OF VPC BY DEMAND PACEMAKER



A. F. Pinnaveer

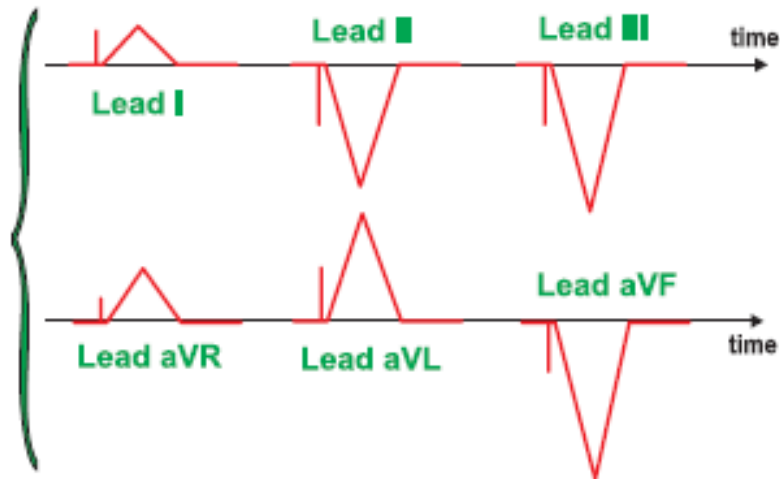
MAGNET APPLICATION ON A DEMAND PACEMAKER



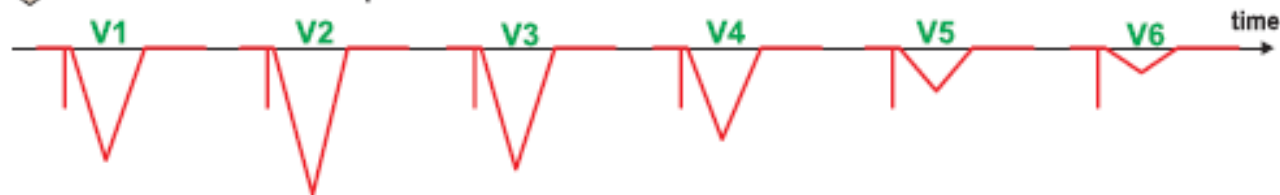
ECG OF RIGHT VENTRICULAR PACING

RV APICAL PACING

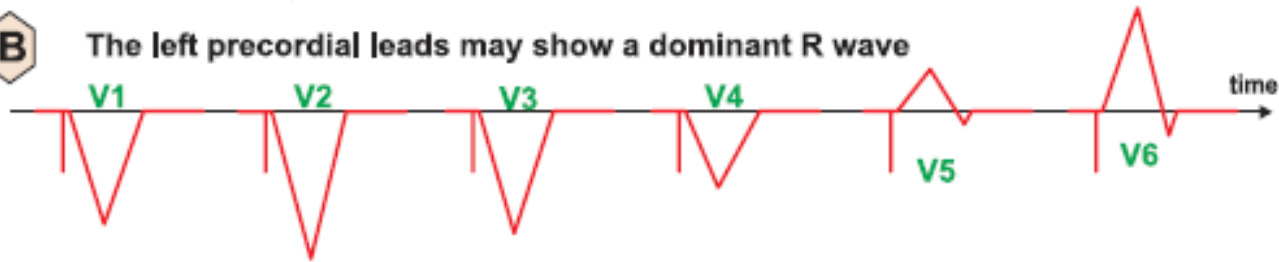
The frontal plane axis is usually left superior. It may also be in the right superior quadrant, where it causes leads I, II & III to be negative and lead aVR to show the largest positive deflection.



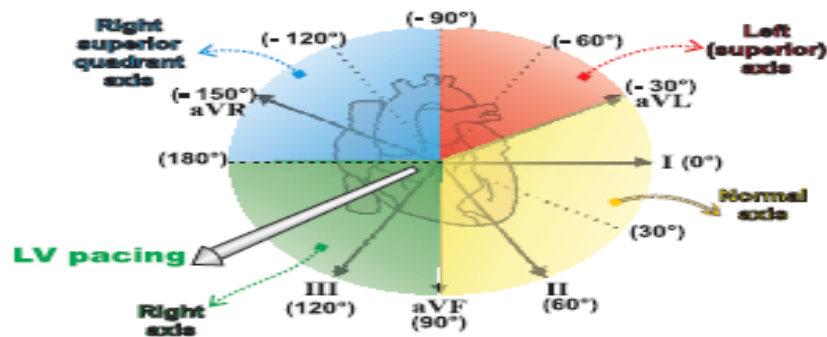
A A typical LBBB pattern in the left precordial leads may not be present and aVL leads show a QS pattern



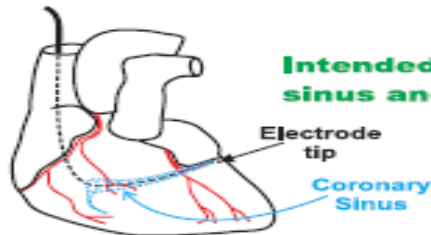
B The left precordial leads may show a dominant R wave



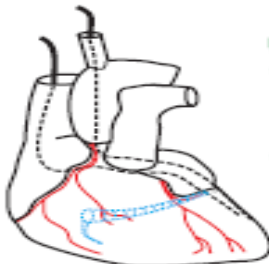
LEFT VENTRICULAR PACING



The mean frontal plane axis of the paced beat is directed to the right lower quadrant (right axis deviation). There is a characteristic tall R wave in lead V1 to at least V3 and often further into the left precordial leads.



Intended LV pacing via coronary sinus and coronary vein

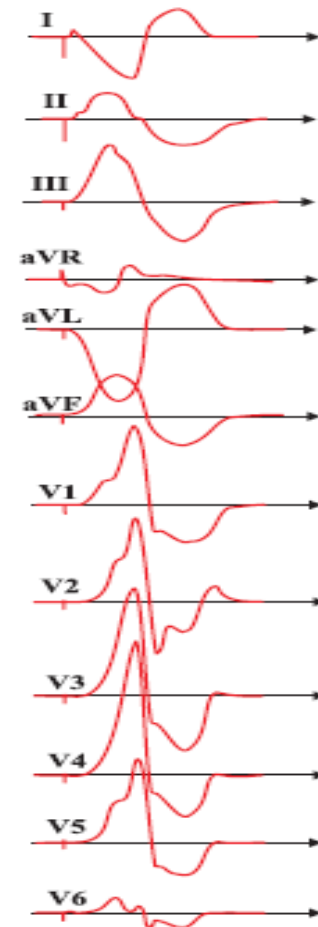


Unintended LV pacing :

- * Passage of lead into LV via patent foramen ovale (from right atrium to left atrium and LV)
- * Via subclavian artery (across the aortic valve) mistaken for the subclavian vein

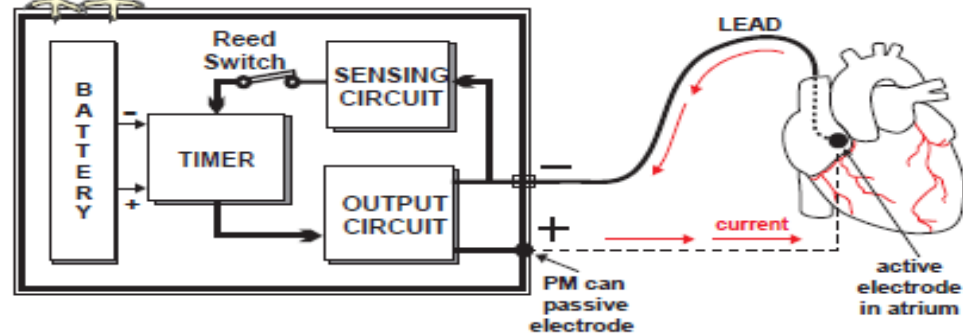
DANGER

A lead within the LV cavity (endocardial site) may cause thrombus formation, cerebral emboli and stroke. The diagnosis of LV endocardial lead misplacement should be suspected if there is a tall R wave at least in leads V1 to V3 and sometimes further in the left-sided precordial leads. The definitive diagnosis requires echocardiography especially by the transesophageal method.

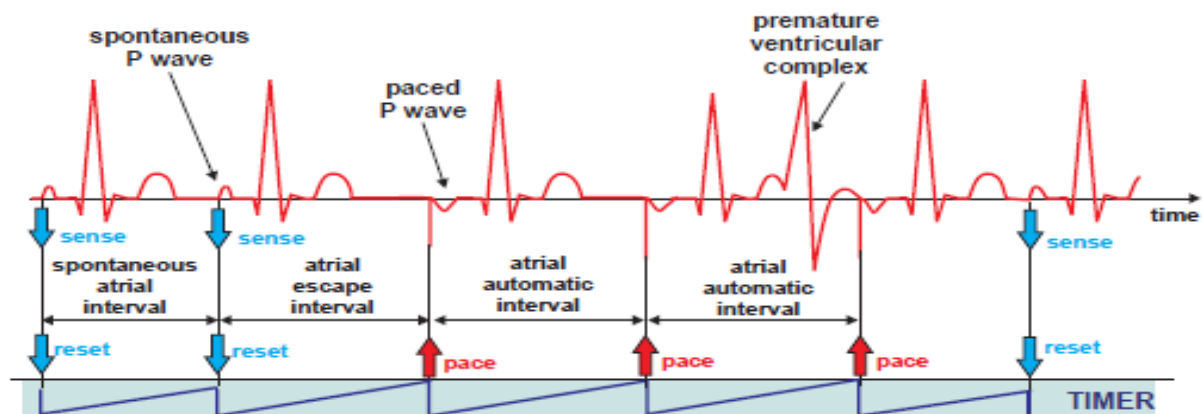




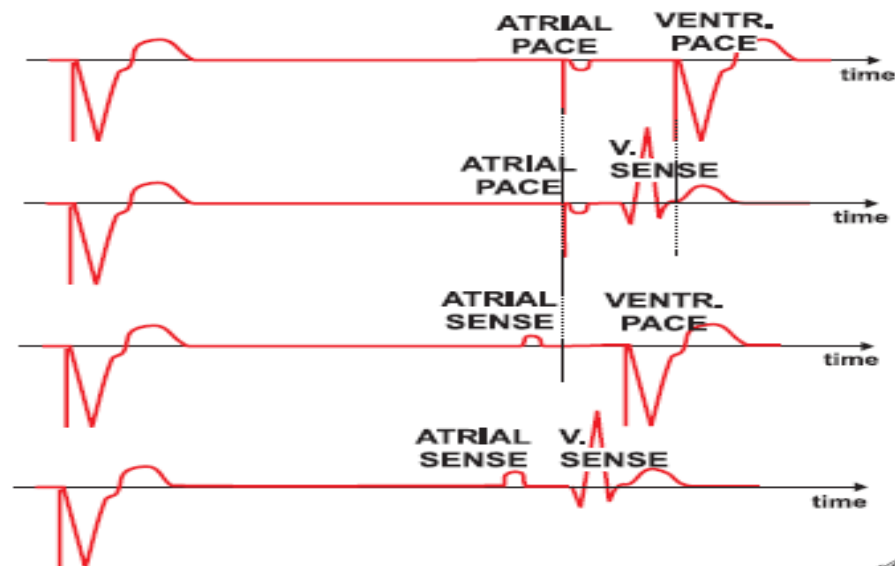
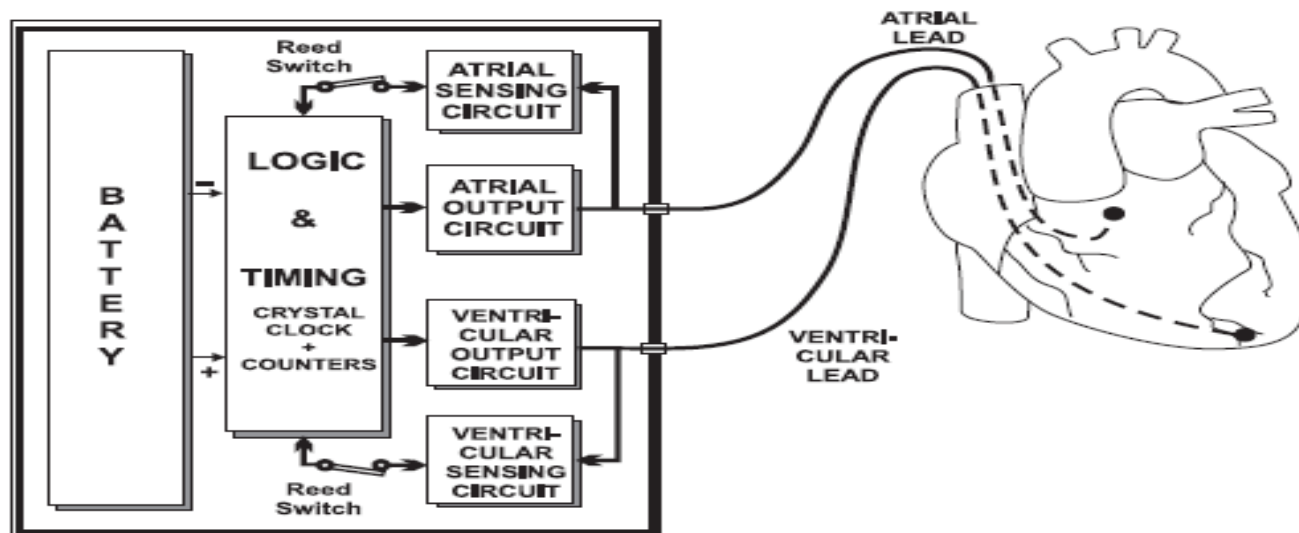
THE AAI PACEMAKER



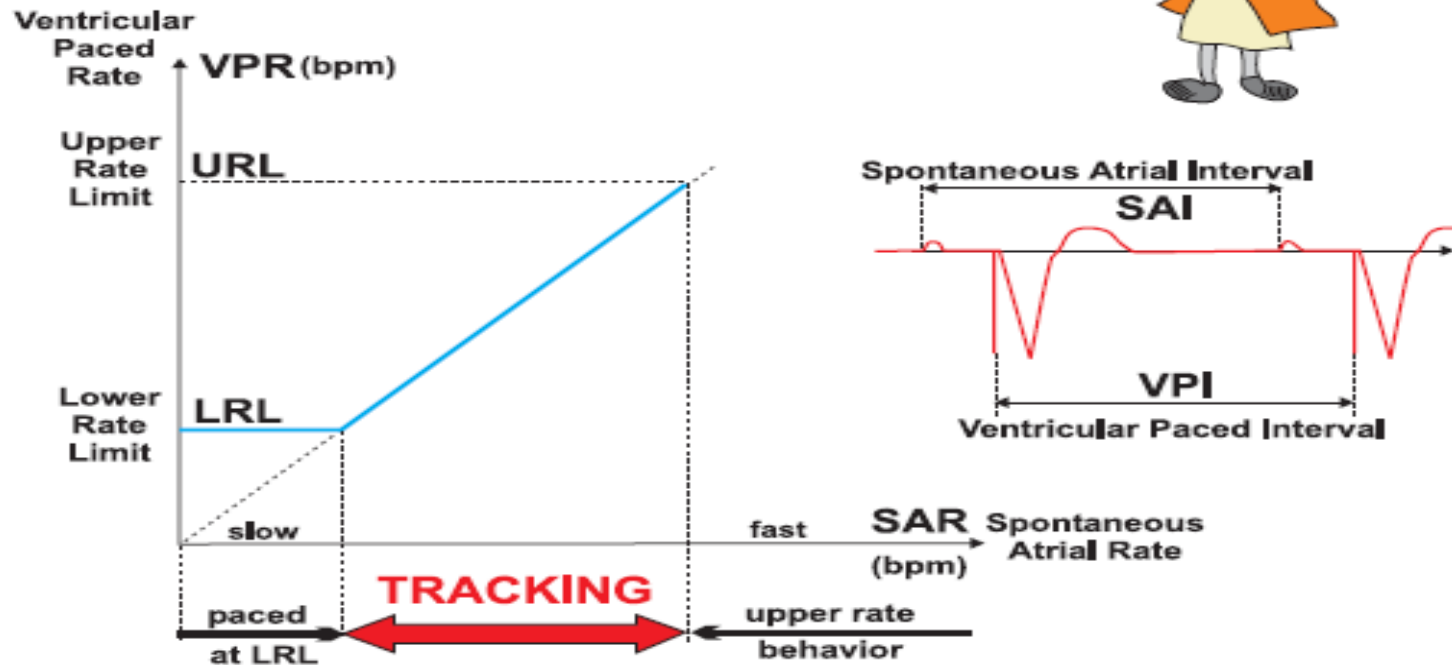
Compared to the VVI pacemaker the **SENSITIVITY** should be higher (i.e. a lower number in mV) because P waves have lower amplitudes than R waves



DUAL CHAMBER PACING



TRACKING is present when the ventricular paced rate follows the spontaneous atrial rate in a 1 : 1 way



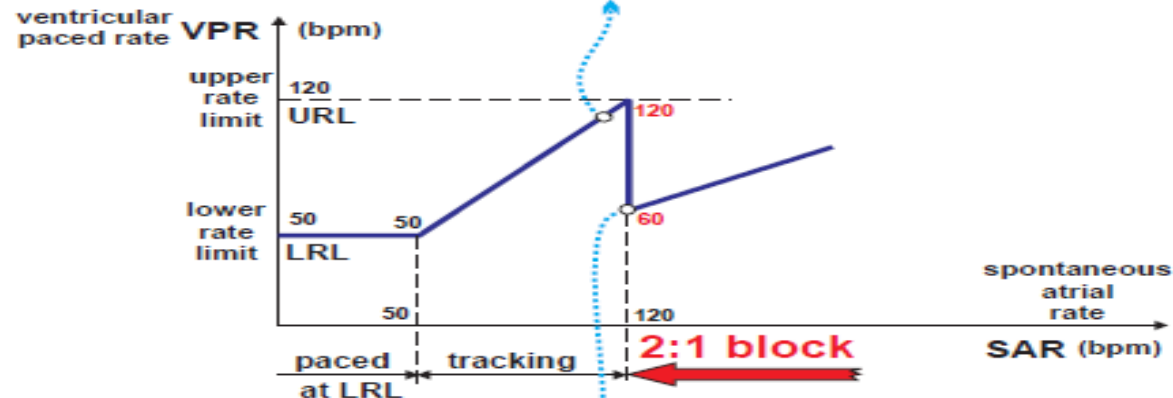
$$\text{Spontaneous Atrial Rate (SAR in bpm)} = \frac{60,000}{\text{Spontaneous Atrial Interval (SAI in ms)}}$$

$$\text{Ventricular paced Rate (VPR in bpm)} = \frac{60,000}{\text{Ventricular Paced Interval (VPI in ms)}}$$

THE TREADMILL STRESS TEST



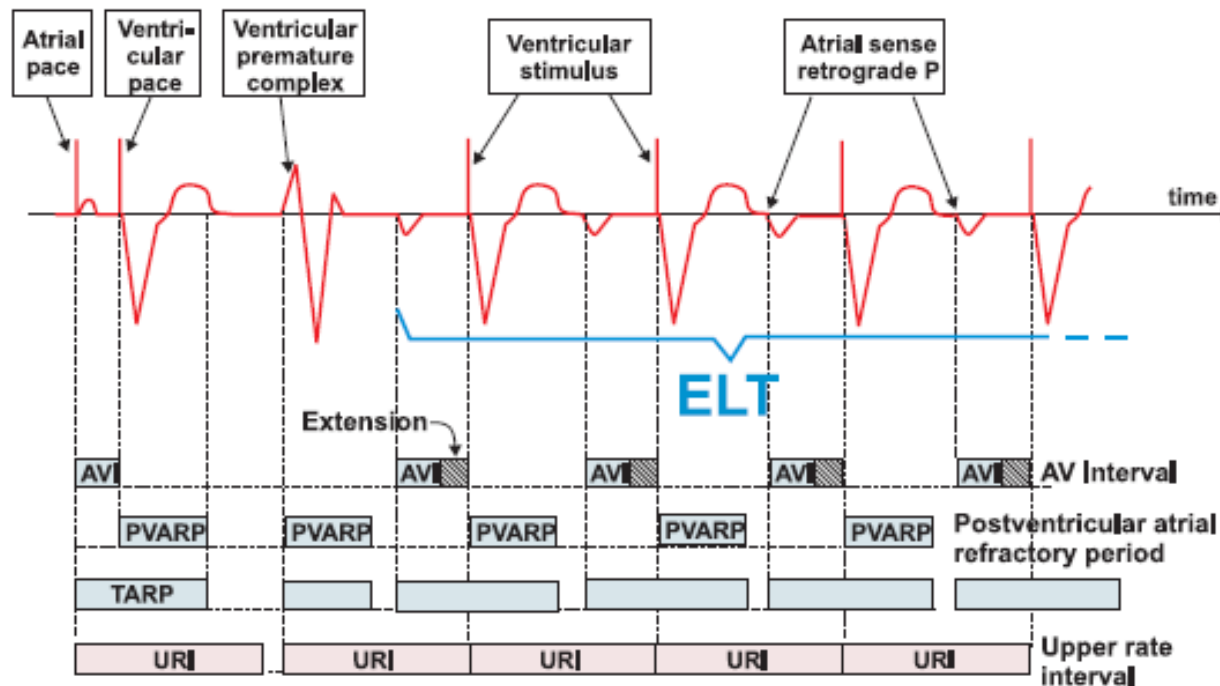
At an atrial rate of 115 bpm and the same paced rate, he still loved his physician....



But...
at an atrial rate of 120 bpm and a paced rate of 60 bpm he felt himself very unhappy
.....

A. F. Pinnave

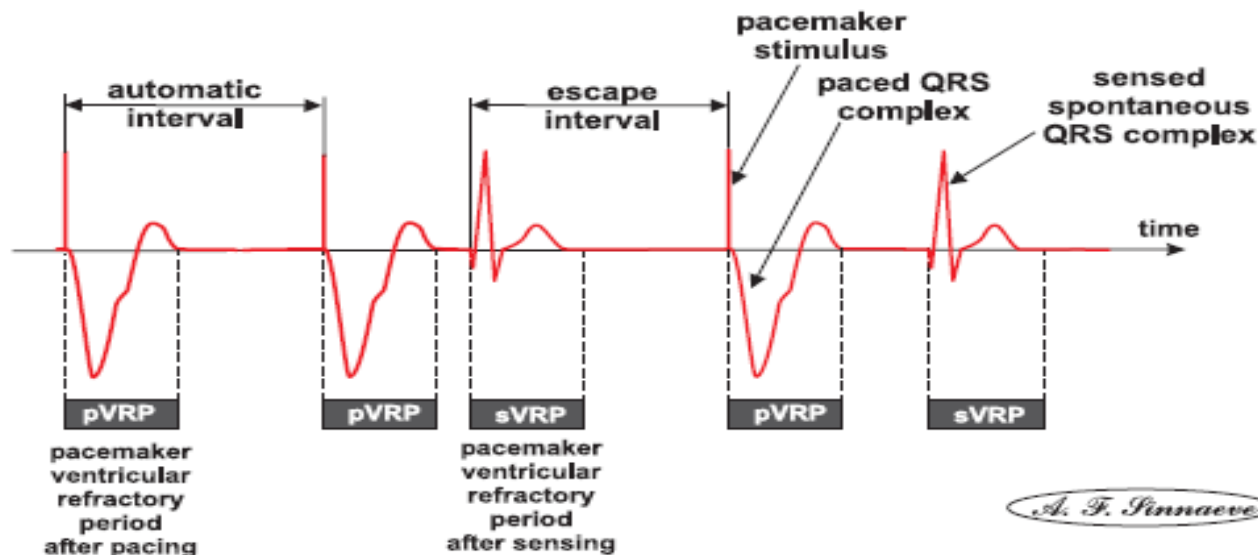
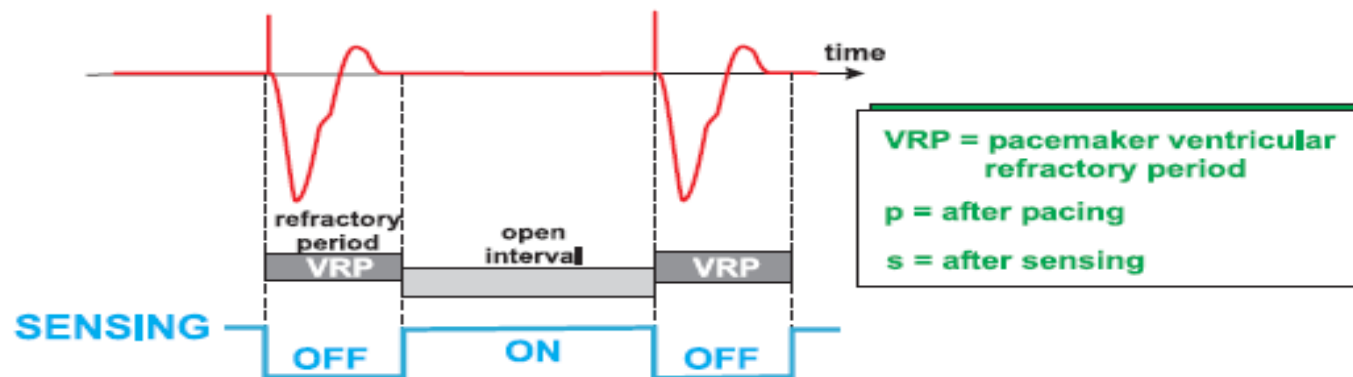
DDD PACEMAKER & ENDLESS LOOP TACHYCARDIA



Endless loop tachycardia often occurs at the upper rate. In such a case as the programmed upper rate interval (URI) is longer than the total atrial refractory interval (TARP), the AV delay is extended to conform to the upper rate interval (URI)



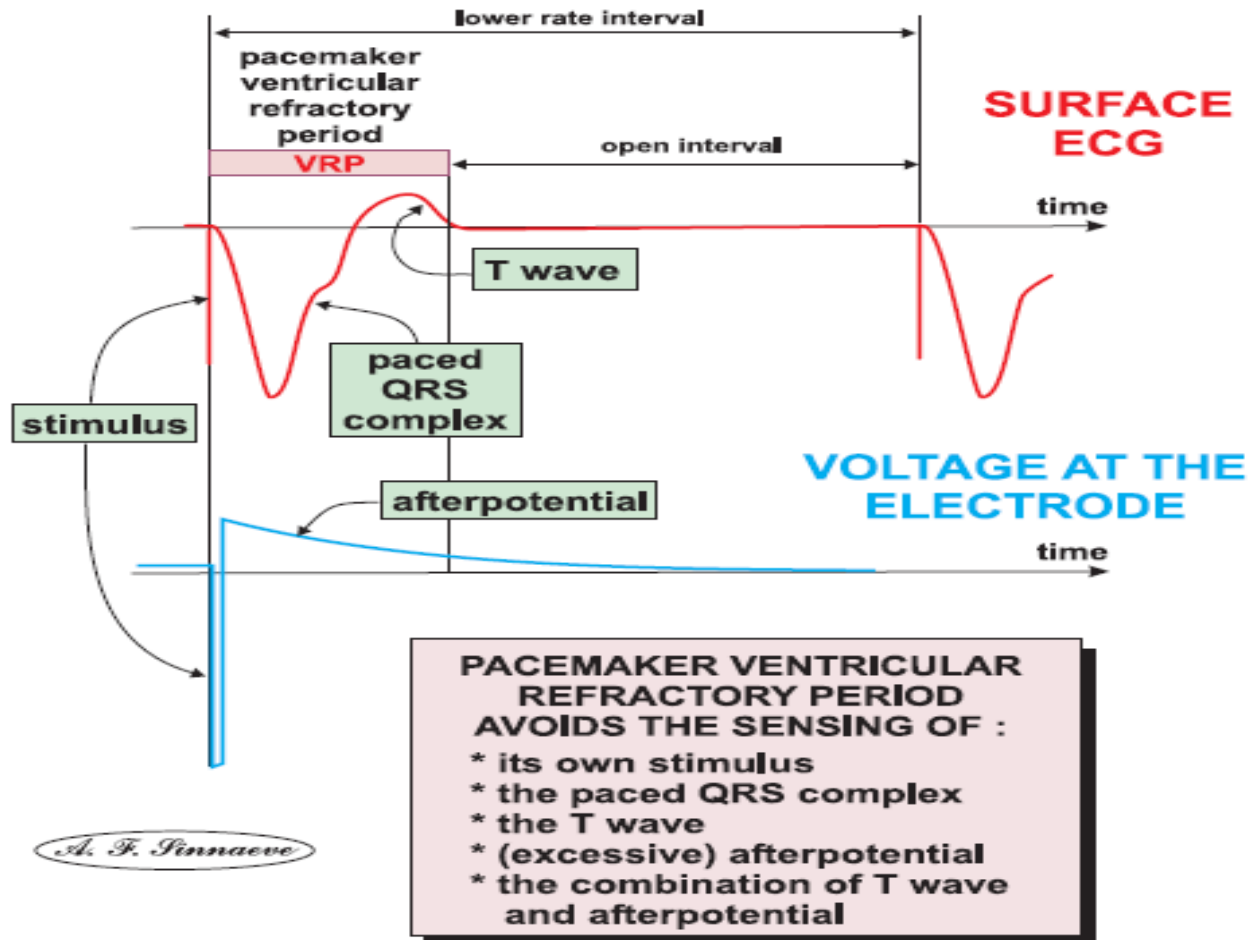
THE PACEMAKER REFRACTORY PERIOD TRADITIONAL CONCEPT



pVRP is often equal to sVRP

A. F. Pinnaeve

FUNCTIONS OF THE PACEMAKER VENTRICULAR REFRACTORY PERIOD



A. F. Pinnaeve

The duration of the pacemaker ventricular refractory period (VRP) is usually 200 - 300 ms

You cannot imagine what **little** details these cardiologists are looking at ...!

RIGHT VENTRICULAR PACING & OLD ANTERIOR MYOCARDIAL INFARCT



A qR or Qr pattern in leads V5 and V6 often indicates an **old** anterior myocardial infarct (MI). This pattern may also occur in leads I and aVL.



A shelf like notch (0.04sec) on the ascending limb of the S wave, called Cabrera's sign, in leads V2 - V5 often indicates an **old** anterior myocardial infarct.

In the case of Cabrera's sign, rule out ventricular fusion beats and retrograde P waves



A. F. Pinnaev

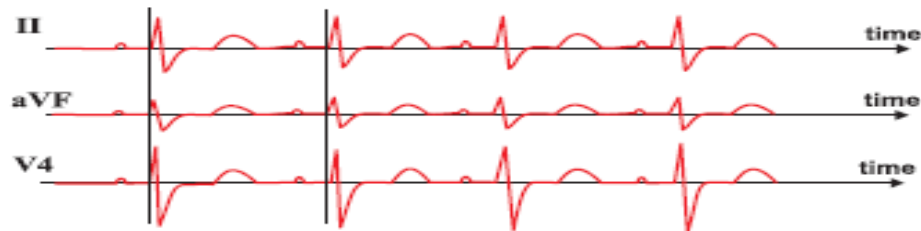
CAUTION

**WATCH
YOUR
STEP**

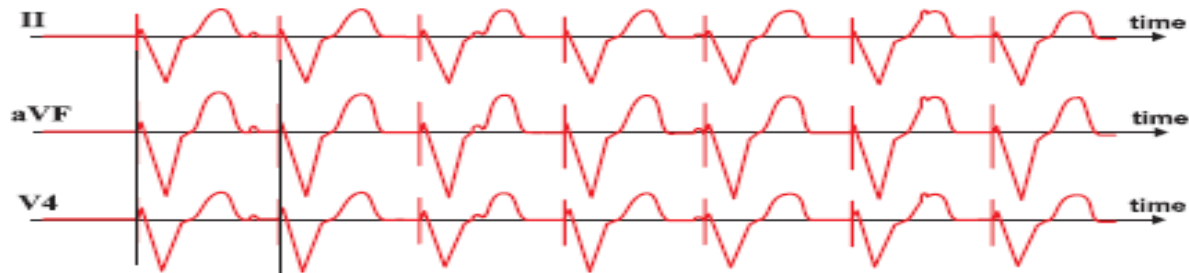
VENTRICULAR PACING & THE MEMORY EFFECT

The underlying ECG cannot be used for the diagnosis of cardiac ischemia because inverted T waves may be due to the memory effect !!!

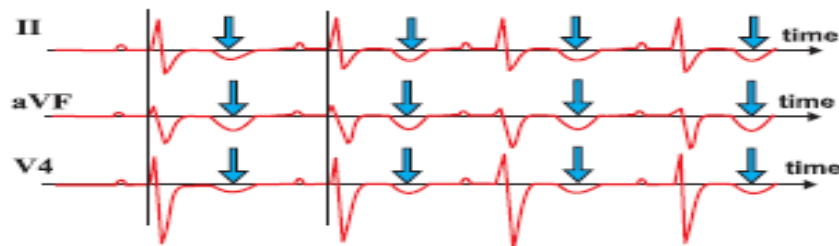
BEFORE PACING



DURING PACING



AFTER PACING



For some time after pacing, the heart seems to remember the abnormal depolarizations. The duration of the memory effect (negative T waves) depends upon the duration of pacing

A. F. Rinaudo



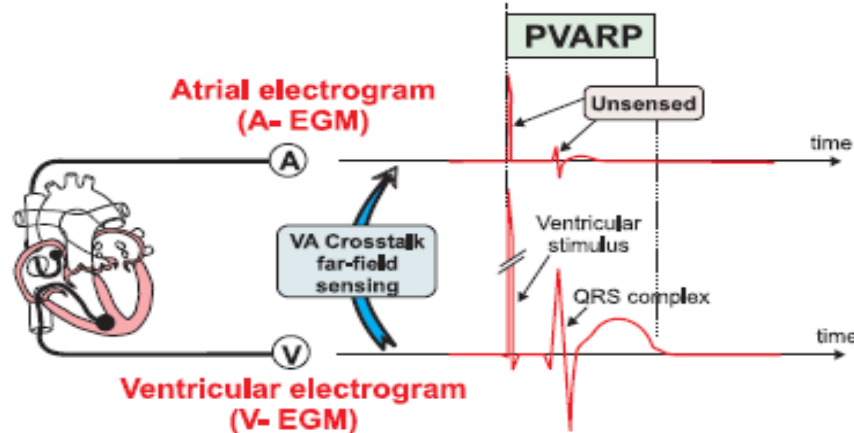
FUNCTIONS OF THE POSTVENTRICULAR ATRIAL REFRACTORY PERIOD (PVARP)

PVARP

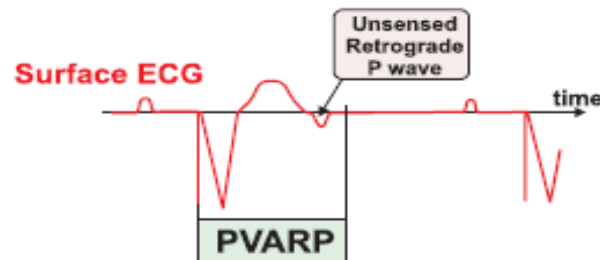
Interval after a ventricular paced or sensed event during which the atrial channel is refractory !!!



1. Avoids the inappropriate atrial sensing of ventricular events (ventricular stimuli, QRS complexes, aberrant T waves)



2. Avoids sensing of retrogradely conducted P waves



A. F. Pinnave

Setting up pacing(TPM)

- ❑ Threshold: set the output to 3V and pace at a rate above the intrinsic cardiac rate.
- ❑ Slowly turn down the box output watching the ECG monitor.
- ❑ Identify the point where capture is lost. The output where the ventricle is recaptured is the pacing threshold.
- ❑ Aim for a threshold of $<1V$.
- ❑ Set output to at least 3x the pacing threshold to ensure a good safety margin.

Setting the box:

- ❑ “output” should be set to 3 times the threshold
- ❑ Set to “demand”
- ❑ “Sensitivity” should be adjusted to ensure that each intrinsic beat is detected but that skeletal muscle interference does not lead to pacemaker inhibition

HARDWARE

748

contraction secondary to abnormal electrical activation disproportionately worsens systolic dysfunction in cardiomyopathy because the remaining myocardium cannot provide the compensatory increase in fiber shortening necessary to maintain stroke volume. Therefore, LV pacing and biventricular (RV and LV) pacing are used to resynchronize ventricular electrical and mechanical activation sequence in patients with symptomatic LV dysfunction.

Hardware

LEADS. The choice of leads for transvenous pacing and defibrillation depends on the patient and application (Fig. 38-5). Unipolar leads sense and pace between a tip electrode and the housing (can) of the pulse generator. Bipolar leads can sense and pace between the tip and ring electrodes (Fig. 38-6). Bipolar sensing is thought to have a lower susceptibility to electromagnetic interference and other far-field signals compared with unipolar sensing. Use of a bipolar lead permits programming of either bipolar or unipolar pacing and sensing.

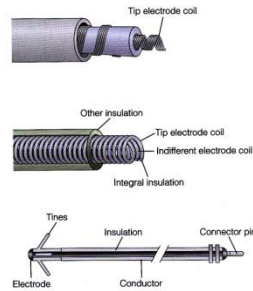


FIGURE 38-5 Basic components of a passive fixation pacing lead. Variants of conductor construction. **Top:** Bipolar coaxial design with an inner multifilar coil surrounded by insulation (inner), an outer multifilar coil, and outer insulation. **Middle:** Individually coated wires wound together in a single multifilar coil for bipolar pacing. **Bottom:** Schematic of passive fixation lead with identification of the electrode, insulation, conductor and connector pin.

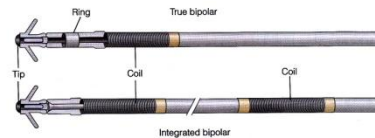


FIGURE 38-6 True bipolar (top) and integrated bipolar (bottom) leads. The true bipolar lead senses between the distal tip and the proximal ring, which are dedicated for pacing and sensing. True bipolar leads have a single coil. In contrast, integrated bipolar leads pace and sense between the tip and the distal coil. The distal coil is used for sensing, pacing, and defibrillation. Integrated bipolar leads also contain a second, proximal coil, increasing the lead surface area for defibrillation.

In contrast, defibrillator leads are either tripolar or quadripolar. All include a pace/sense tip electrode and a RV coil electrode. True bipolar leads sense and pace between the tip electrode and a closely spaced dedicated ring electrode. Integrated bipolar sensing leads use the RV defibrillation coil as the anodal ring for sensing and pacing. Dual-coil defibrillation leads have a proximal defibrillation coil intended to rest in the superior vena cava when the tip is in the RV apex. Most ICD systems shock from the RV coil to the pulse generator can and the superior vena cava coil.

For most patients, dual- or single-coil and true or integrated bipolar systems are effective. Single-coil systems may be favored in younger patients who have a potential need for future extraction or as an additional lead in patients with a preexisting dual-coil lead to minimize lead-lead interactions. True bipolar sensing is preferred in pacemaker-dependent patients, in the setting of abandoned leads, and for patients who may be at increased risk for exposure to electromagnetic interference. Dual-coil systems may be preferable when higher DFTs are anticipated (hypertrophic cardiomyopathy, arrhythmic therapy, some inherited sodium channel abnormalities), although clinical predictors of high DFTs have been limited, and less than 5% of patients require system revision.¹

Nontransvenous, epicardial (myocardial) leads can be necessary in patients with congenital cardiac anomalies associated with univentricular hearts or that prevent the access needed for transvenous leads as well as in patients with tricuspid valve conditions that preclude lead placement across the valve (e.g., mechanical prosthetic valve). However, coronary venous pacing can obviate the need for some epicardial pacing. Historically, epicardial leads have had higher pacing thresholds and more conductor failures than transvenous leads have.

PULSE GENERATOR COMPONENTS. All pulse generators include a battery, most commonly lithium-iodine in pacemakers and lithium-silver vanadium oxide in ICDs. Located adjacent to the battery is the "hybrid" or circuit board, which is the mini-computer that processes bidirectional information between incoming signals from the heart and the programmer and responds to cardiac signals as programmed. The hybrid also stores information that can be retrieved via the programmer. An ICD also includes a high-voltage transformer and a bulky system of high-voltage capacitors that allow the device to develop and to store the charge adequate for defibrillation.

Sensing and Detection

Delivery of appropriate electrical therapy depends on sensing of cardiac depolarizations and detection of arrhythmias by analysis of the timing and morphology of sensed events. When a depolarization wave front passes the tip electrode of an intracardiac lead, a deflection in the continuous electrogram (ECG) signal travels instantaneously via the electrode to the pulse generator. There, the signal is amplified, filtered, digitized, and processed by the sensing electronics. A sensed event is an instant in time when the device determines that an atrial or ventricular depolarization has occurred on the basis of processing the continuous ECG signal. Detection algorithms process sensed events to classify the atrial or ventricular rhythm. This classification is used to control beat-by-beat paced events, to change the pacing mode in response to a pathologic atrial rhythm, to store data about untreated tachyarrhythmias, and to treat tachyarrhythmias with antitachycardia pacing or shocks.

Intracardiac Electrogram

An ECG displays the electrical potential difference between two points in space over time. The electrocardiogram (ECG), recorded from

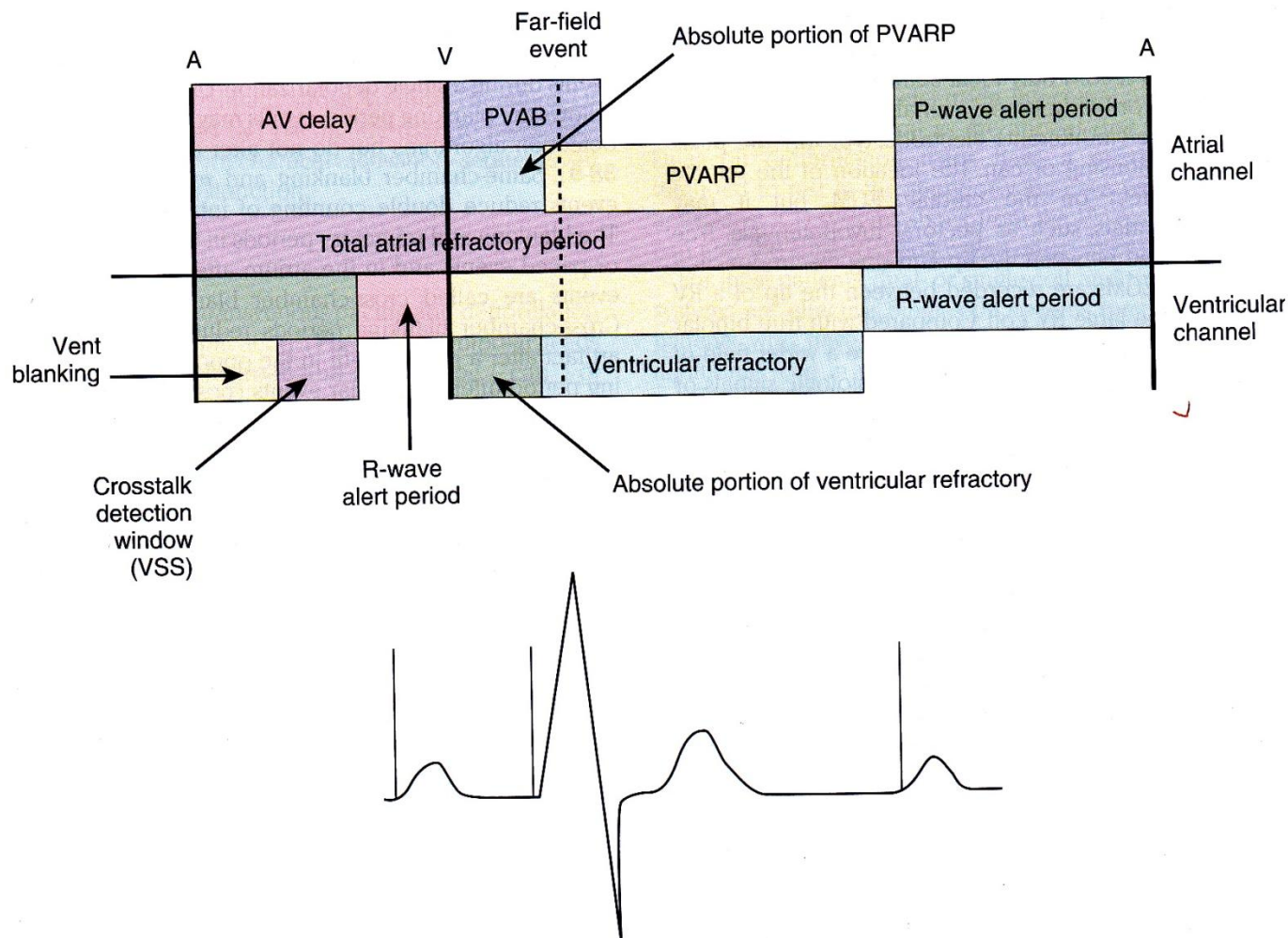


FIGURE 38-9 Schematic representation of the timing cycle interactions of most refractory and blanking periods available on contemporary dual-chamber pacemakers. PVAB, postventricular atrial blanking; PVARP, postventricular atrial refractory period.

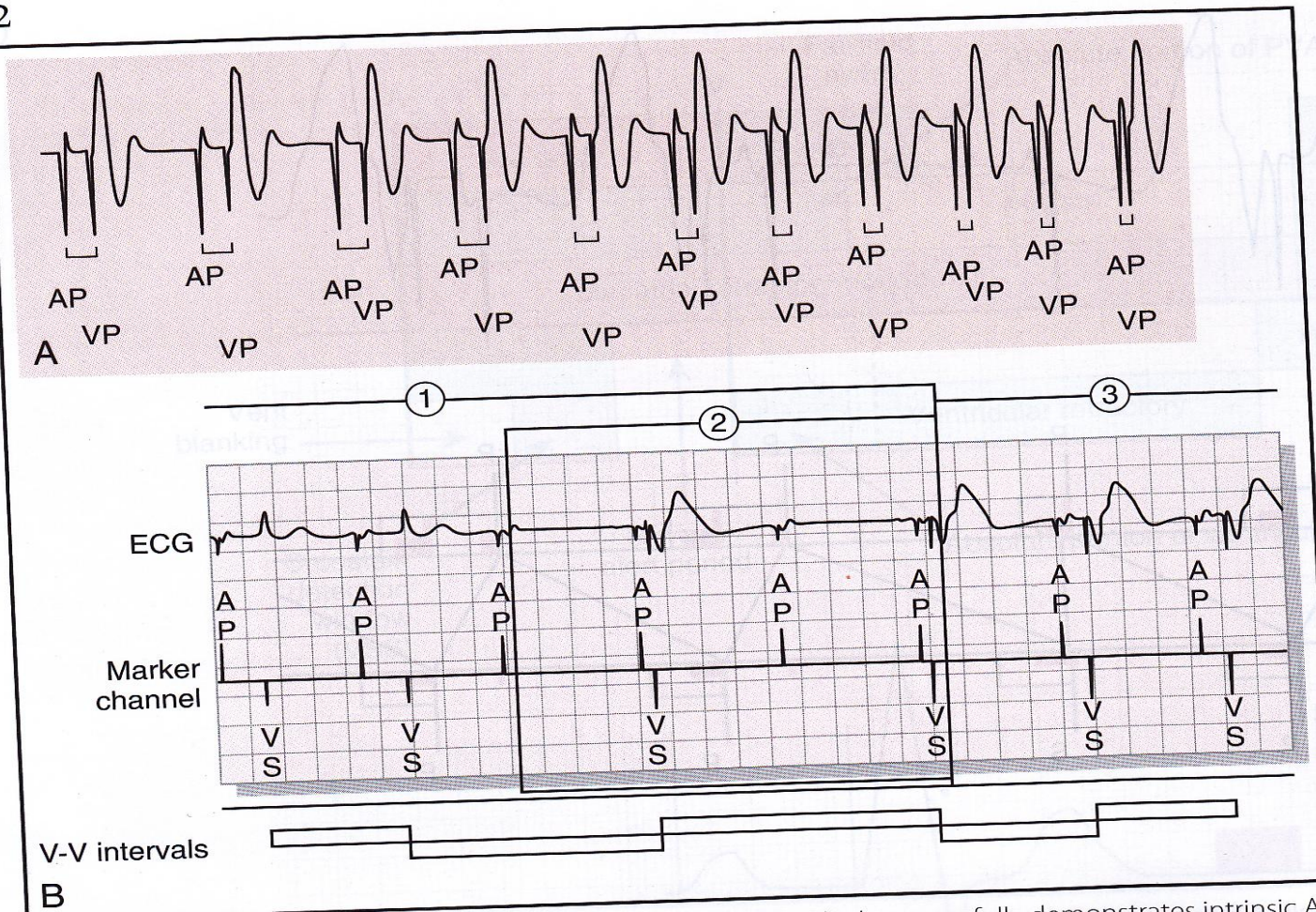


FIGURE 38-13 **A**, Schematic example of AV search hysteresis, which successfully demonstrates intrinsic AV conduction. **B**, Example of managed ventricular pacing. Initially, AAIR pacing is seen; if an atrial pace event occurs without a ventricular sensed event, a ventricular backup output occurs and the pacemaker then switches to DDDR mode.

FIGURE 38-9 Schematic representation of the timing cycle for a form of most refractory and blanking. The cycle includes: Atrial blanking, VVAA postventricular atrial refractory period.

TABLE 38-1 NASPE/BPEG Generic Code for Antibradycardia Pacing

POSITION	I	II	III	IV	V
Category	Chamber(s) paced O = None A = Atrium V = Ventricle D = Dual (A + V)	Chamber(s) sensed O = None A = Atrium V = Ventricle D = Dual (A + V)	Response to sensing O = None T = Triggered I = Inhibited D = Dual (T + I)	Rate modulation O = None R = Rate modulation	Multisite pacing O = None A = Atrium V = Ventricle D = Dual (A + V)
Manufacturers' designation only	S = Single (A or V)	S = Single (A or V)			

See text for explanation of use of the code.

BPEG = British Pacing and Electrophysiology Group; NASPE = North American Society of Pacing and Electrophysiology.

From Bernstein AD, Daubert JC, Fletcher RD, et al: The revised NASPE/BPEG generic code for antibradycardia, adaptive-rate, and multisite pacing. *Pacing Clin Electrophysiol* 25:260, 2002.

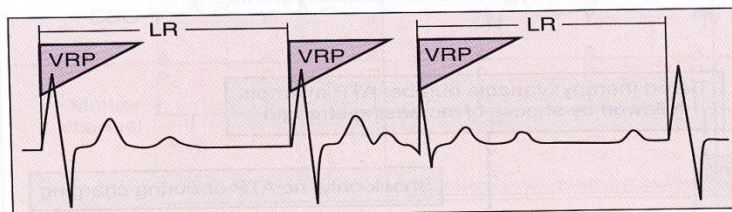


FIGURE 38-17 The VVI timing cycle consists of a defined lower rate (LR) limit and a ventricular refractory period (VRP, represented by triangle). When the LR limit timer is complete, a pacing artifact is delivered in the absence of a sensed intrinsic ventricular event. If an intrinsic QRS occurs, the LR limit timer is started from that point. A VRP begins with any sensed or paced ventricular activity.

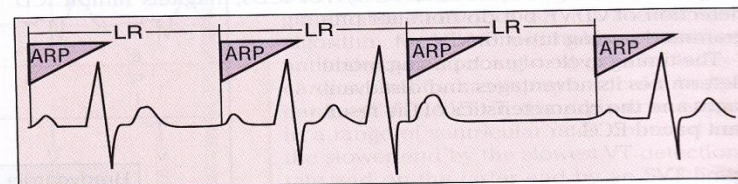


FIGURE 38-18 The AAI timing cycle consists of a defined lower rate (LR) limit and an atrial refractory period (ARP). When the LR limit timer is complete, a pacing artifact is delivered in the atrium in the absence of a sensed atrial event. If an intrinsic P wave occurs, the LR limit timer is started from that point. An ARP begins with any sensed or paced atrial activity. In the AAI mode, only atrial activity is sensed. In this example, it may appear unusual for paced atrial activity to occur so soon after intrinsic ventricular activity. Because sensing occurs only in the atrium, ventricular activity would not be expected to reset the pacemaker's timing cycle. (From Hayes and Levine. By permission of Blackwell Scientific Publications.)

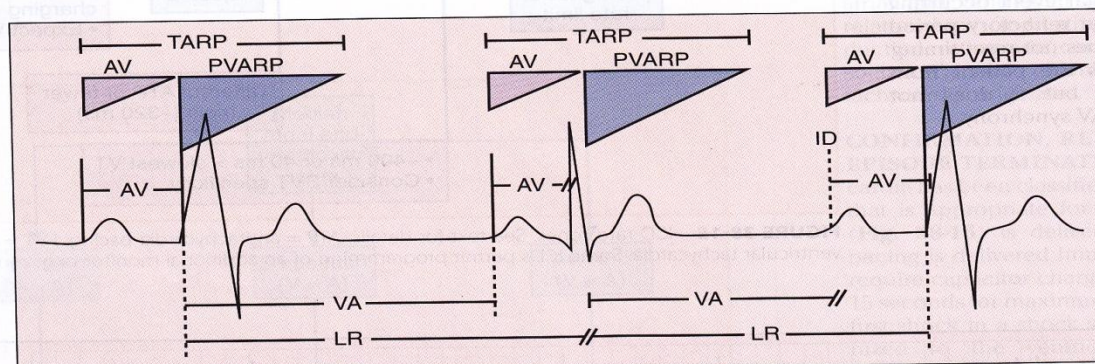


FIGURE 38-19 The timing cycle in DDD consists of a lower rate (LR) limit, an atrioventricular (AV) interval, a ventricular refractory period, a postventricular atrial refractory period (PVARP), and an upper rate limit. If intrinsic atrial and ventricular activity occurs before the LR limit times out, both channels are inhibited and no pacing occurs. In the absence of intrinsic atrial and ventricular activity, AV sequential pacing occurs (first cycle). If no atrial activity is sensed before the ventriculoatrial (VA) interval is completed, an atrial pacing artifact is delivered, which initiates the AV interval. If intrinsic ventricular activity occurs before the termination of the AV interval, the ventricular output from the pacemaker is inhibited, that is, atrial pacing (second cycle). If a P wave is sensed before the VA interval is completed, output from the atrial channel is inhibited. The AV interval is initiated, and if no ventricular activity is sensed before the AV interval terminates, a ventricular pacing artifact is delivered, that is, P-synchronous pacing (third cycle). ID = intrinsic deflection; TARP = total atrial refractory period.

Troubleshooting

- ❑ 1.Failure to capture
- ❑ 2.Failure to pace or output
- ❑ 3.Undersensing
- ❑ 4.Oversensing
- ❑ 5.Pacing at a rate not consistent with the programmed rate

Failure to capture

- ❑ Pacing stimulus without subsequent cardiac depolarization
- ❑ It may be related to the pacing system, the patient, or patient-system interaction.
- ❑ Stimulus occurs in the physiologic refractory period of a depolarization

LOSS OF VENTRICULAR CAPTURE BY VISIBLE PACEMAKER STIMULI



1 FUNCTIONAL

- ✓ Normal situation : stimuli in myocardial refractory period.

2 ELECTRODE-TISSUE INTERFACE

LEAD DISPLACEMENT

- ✓ Early displacement or unstable position of pacing leads (commonest cause).
- ✓ Malposition into the coronary venous system.
- ✓ Twiddler's syndrome causing late displacement.
- ✓ Perforation of right ventricle by ventricular lead.

NO APPARENT LEAD DISPLACEMENT

- ✓ Microdislodgment (a diagnosis of exclusion) causes a marked rise in capture threshold but displacement is not apparent on a chest x-ray.
- ✓ Elevated pacing threshold without obvious lead displacement (exit block) : Acute or chronic reaction at the electrode-tissue interface.
- ✓ Subcutaneous emphysema.
- ✓ Myocardial infarction or ischemia, hypoxia.
- ✓ Hypothyroidism.
- ✓ Elevation of pacing threshold after defibrillation or cardioversion. This is usually transient for a few minutes or less.
- ✓ Electrolyte abnormalities usually hyperkalemia, severe acidosis.
- ✓ Drug effect : Flecainide and propafenone can elevate the pacing threshold with therapeutic doses.

3 ELECTRODE

- ✓ Fracture, short circuit or insulation break.

4 PULSE GENERATOR

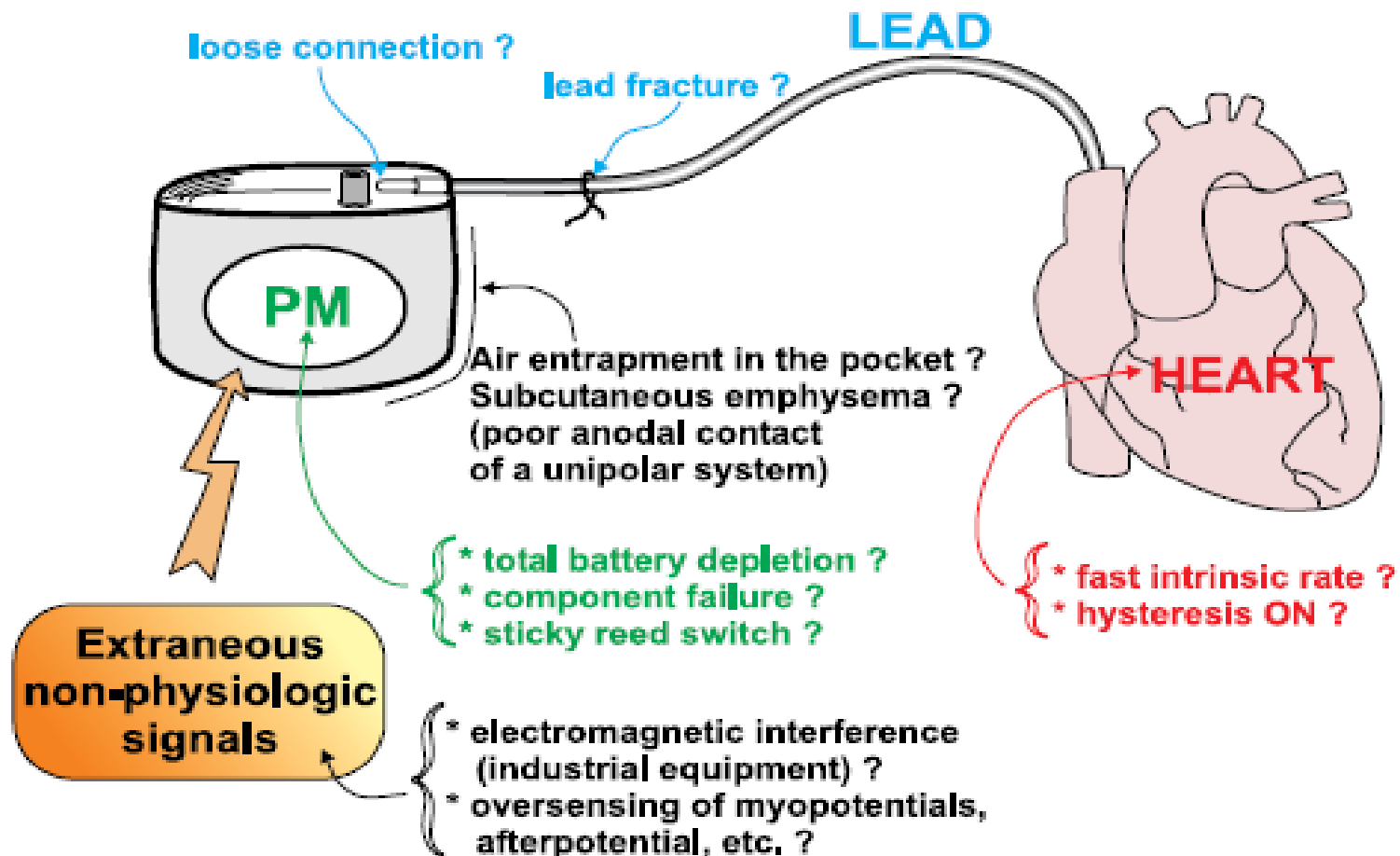
- ✓ Normal pacemaker with incorrect programming of parameters.
- ✓ Pacemaker failure from exhaustion or component failure.
- ✓ Iatrogenic causes : Component failure after defibrillation, electrocautery and therapeutic radiation.

A. F. Pinnaeve

Failure to pace

- Is most commonly due to oversensing of physiologic or nonphysiologic signals, resulting in inhibition of the pacing output
- It may be caused by failure of the pulse generator or an open circuit(A lead fracture or A loose set screw)

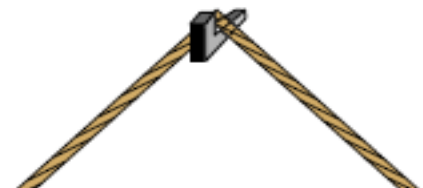
CAUSES OF MISSING STIMULI DURING VVI PACING



ANALYSIS OF LEAD PROBLEMS



Be cunning as a fox ! You can get a lot of information about the leads, just by looking at the pacing impedance and the voltage threshold. The secret is to look at both !



	IMPEDANCE	VOLTAGE THRESHOLD
NORMAL LEAD PLACEMENT	NORMAL	NORMAL
LEAD DISPLACEMENT OR EXIT BLOCK	NORMAL	HIGH
LEAD FRACTURE	HIGH	HIGH
LEAD INSULATION DEFECT	LOW	MAY BE MODERATELY INCREASED

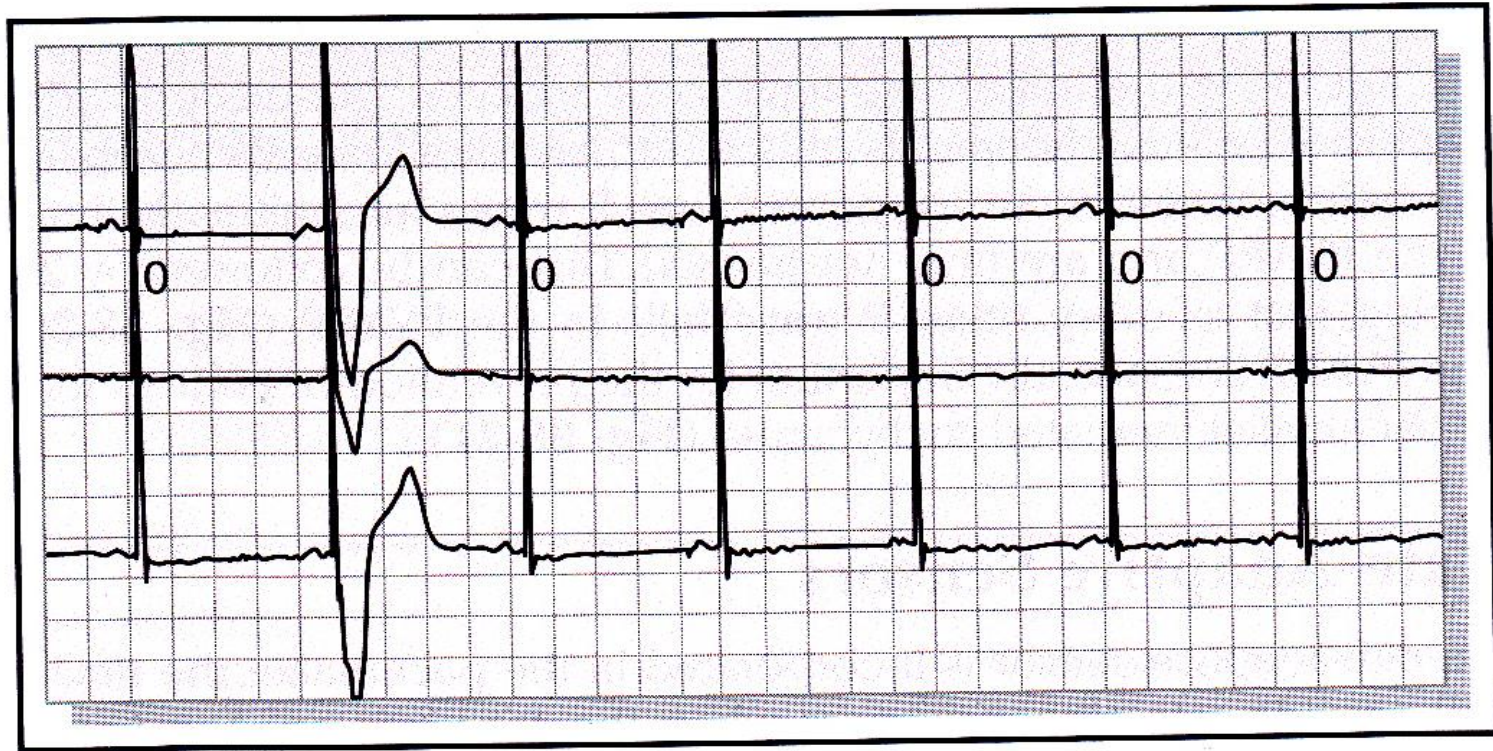


FIGURE 38-22 Three-channel tracing from an ambulatory monitor that was obtained because the patient had recurrent symptoms after pacemaker implantation. The tracing demonstrates intermittent failure to capture, and the pacemaker malfunction correlated with symptoms. The patient was found to have developed excessively high pacing thresholds on epicardial pacing leads.

FIGURE 38-11 Electrocardiographic example of crosstalk. Although crosstalk appears to be the most likely cause of ventricular failure to output when the surface ECG is assessed, it is confirmed by the telemetered ladder diagram. The ladder diagram, a type of diagnostic channel that is not commonly seen with contemporary devices, confirms that the atrial output was detected almost simultaneously on the ventricular sensing channel and that ventricular output was inhibited. P, paced; S, sensed.

Crosstalk

- Is a specific form of oversensing in which the atrial pacing stimulus is sensed on the ventricular channel, resulting in inhibition of ventricular output

Pacing at a rate not consistent with the programmed rate

- ▣ Pacing with a shorter than expected escape interval indicates undersensing of myocardial depolarization

Failure to Respond to Resynchronization Pacing

- ❑ Caused by:-- Patient-related factors
- ❑ -- System-related Factors
- ❑ -- Interaction between the patient and the system

Patient-related

- ▣ Resynchronization of less than 90% of R-R intervals caused by conducted AF or frequent PVC,s.

System-related:

- ❑ Loss of LV capture due to lead dislodgement

- ❑ Patient-system interaction:

Placement of the LV lead at an ineffective site for resynchronization and chronic changes in pacing threshold that can occur in any pacing system

Implant-related complications

- ❑ Hematoma
- ❑ Traumatic pneumothorax
- ❑ Inadvertant arterial puncture
- ❑ Air embolism
- ❑ A-V fistula
- ❑ Thoracic duct injury
- ❑ Subcutaneous emphysema
- ❑ Brachial plexus injury
- ❑ LV lead
- ❑ Tamponad (acute- asymptomatic)

Lead –related complications

- ❑ Dislodgment
- ❑ Header-connector pin problems
- ❑ conductor/cable(lead) fracture
- ❑ Insulation break

Complications of pacemakers

- Two major groups of complications are associated with pacemaker implantation:
 - (a) nonelectrical complications including acute complications at the time of implantation such as pneumothorax and complications of lead placement and pocket formation;
 - (b) electrical complications.

Nonelectrical complications

Table 7. Nonelectrical or arrhythmic complications.

Venous access	Pneumothorax Hemothorax Air embolism Brachial plexus injury Thoracic duct injury Trauma to the subclavian artery Hematoma
Pacemaker pocket	Infection, septicemia, etc. Conservative therapy is often unsuccessful and removal of the entire system may be required Hematoma/seroma Erosion Pacemaker migration Twiddler's syndrome Muscle stimulation from either a flipped but normally functioning unipolar or extravascular insulation defect Chronic pain including subcuticular malposition of the pulse generator
Intravascular	Subclavian or innominate vein thrombosis Thrombosis of superior vena cava Coronary sinus dissection or perforation during implantation of a left ventricular lead Large right atrial thrombus Endocarditis with vegetations Manifest pulmonary embolism (rare) Cardiac perforation Cardiac tamponade Entanglement of lead in the tricuspid valve and ruptured chordae Tricuspid insufficiency Pericardial rub
Lead problems	Displacement Malposition in the coronary venous system Endocardial left ventricular malposition across a patent foramen ovale or via subclavian arterial puncture (or via atrial or ventricular septum defect) Right ventricular perforation or lead perforation of the interventricular septum Diaphragmatic pacing. Left side with or without right ventricular perforation and right side by phrenic nerve stimulation by atrial pacing Intercostal muscle stimulation due to right ventricular perforation Post pericardiotomy syndrome (pericarditis etc.) with or without lead perforation Intracardiac rupture of lead during attempt to remove old or broken lead